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Southern Medical Association, Thirty-Fifth Annual Meeting, St. Louis, Missouri, November 10-13

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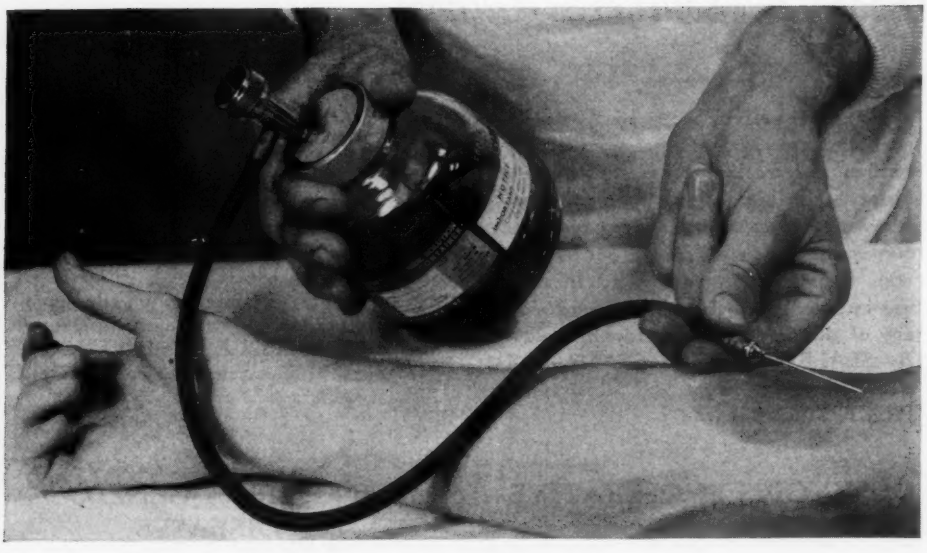

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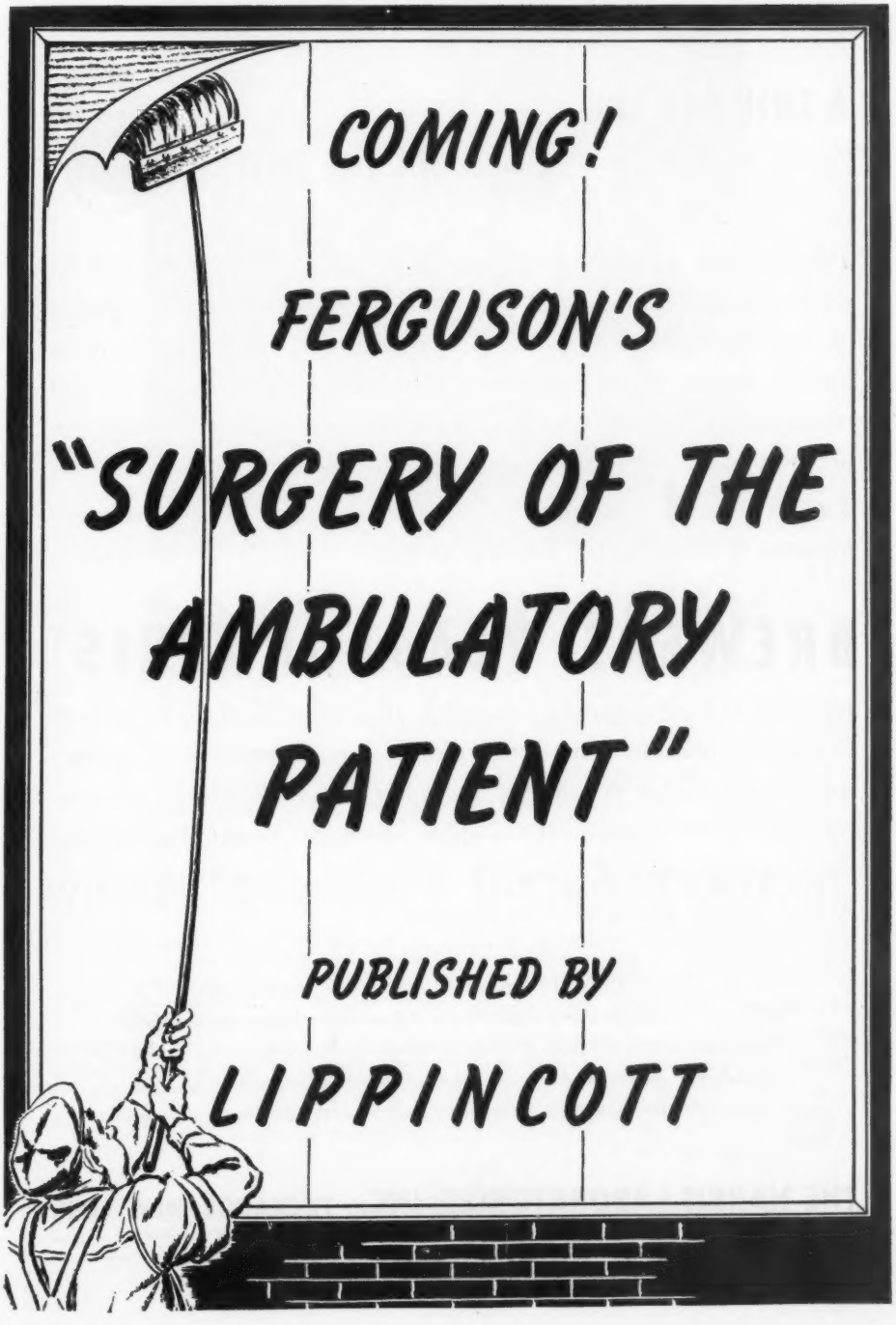
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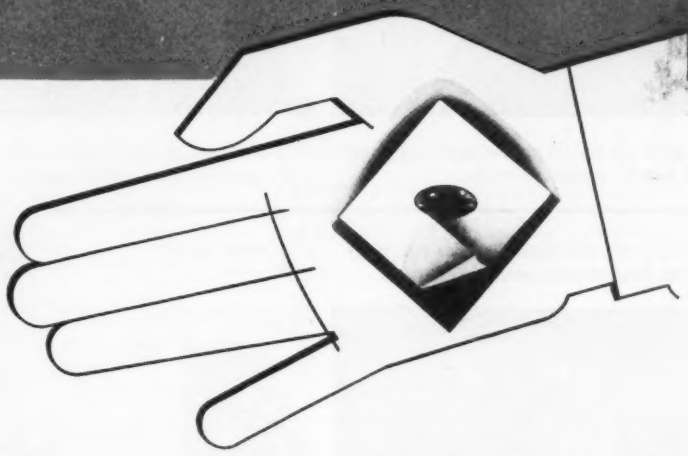

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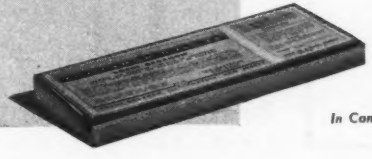

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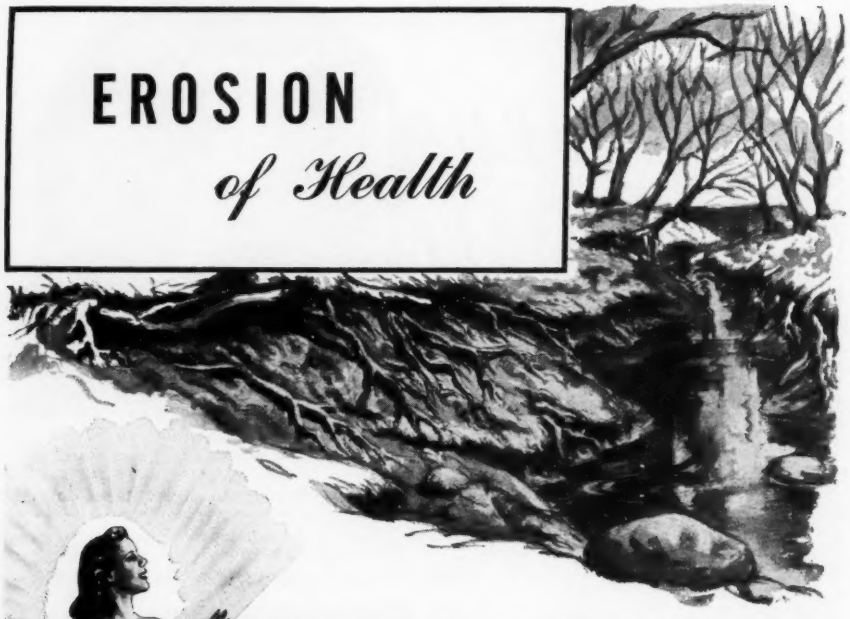

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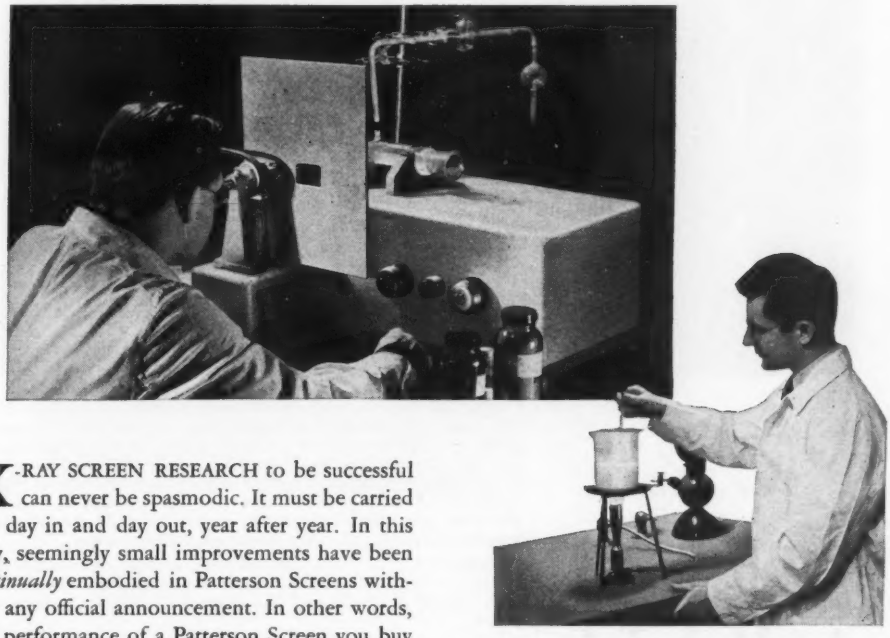

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1. Endocrines in Obstetrics and Gynecology, Raphael Kurzrok, Williams & Wilkins Co., 1937.

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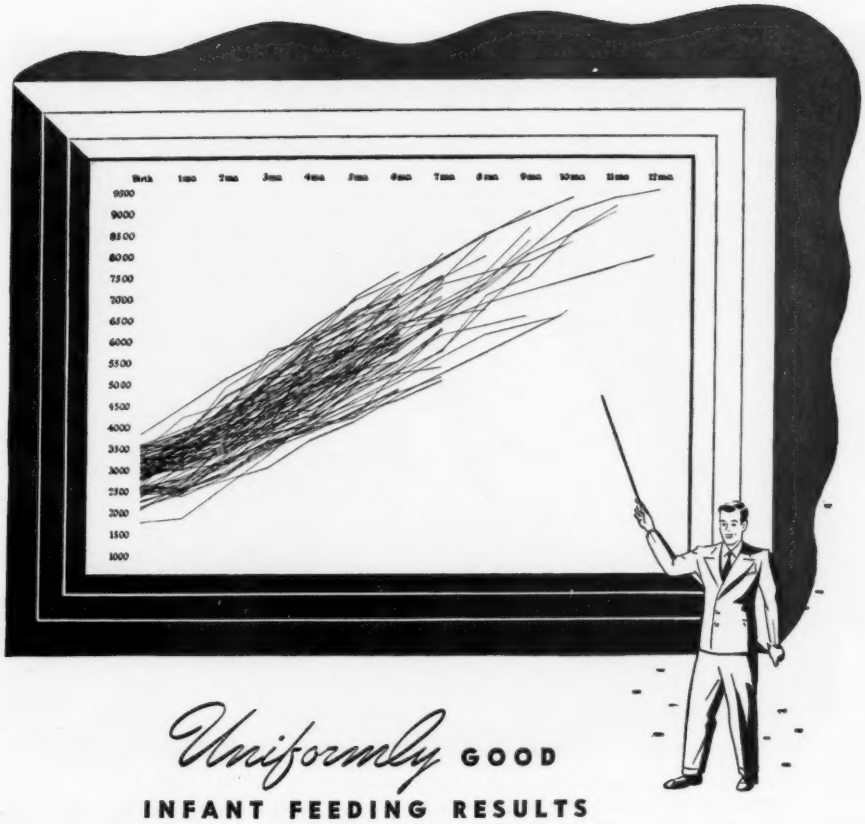

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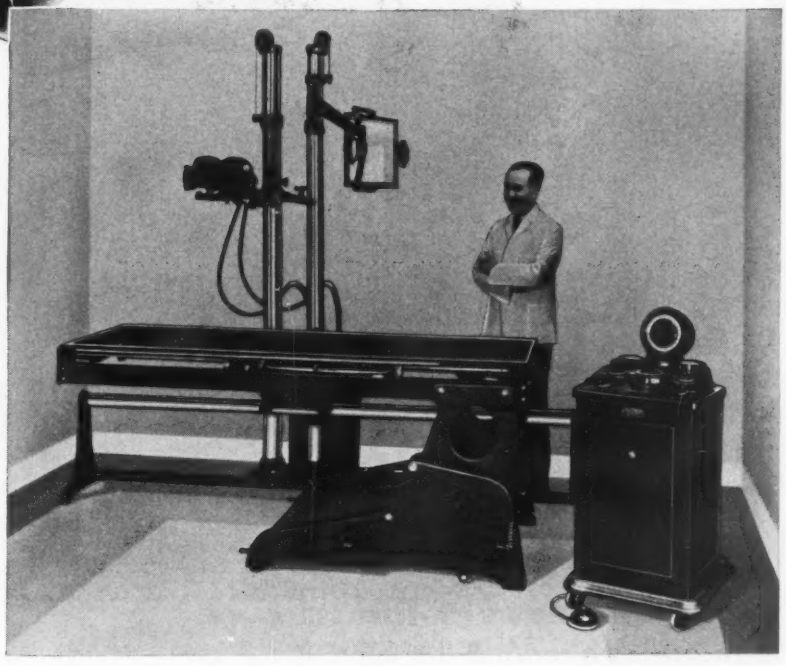

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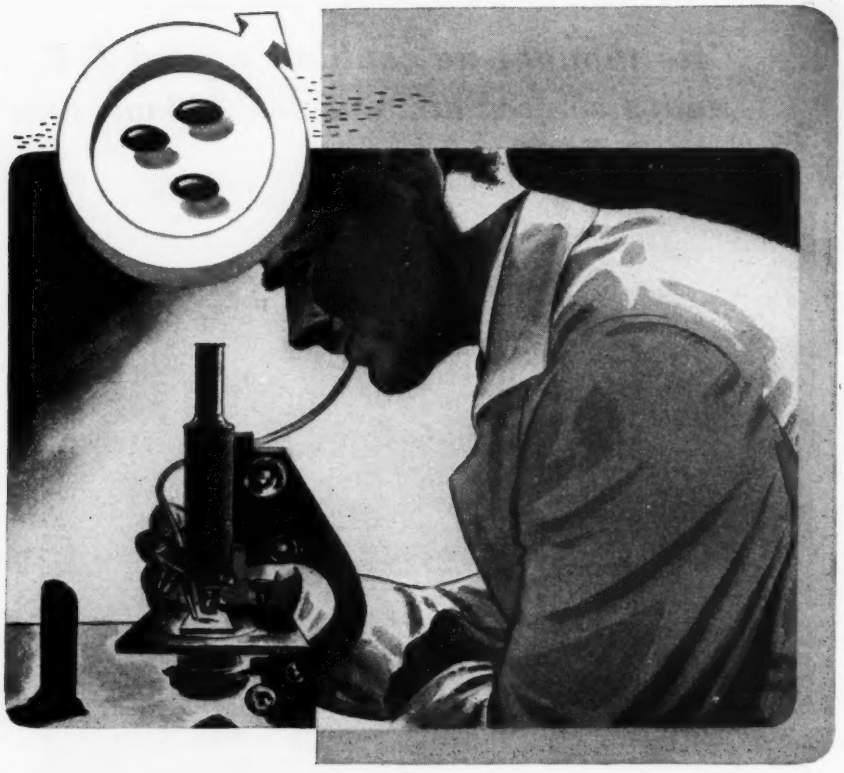

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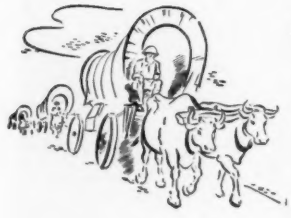

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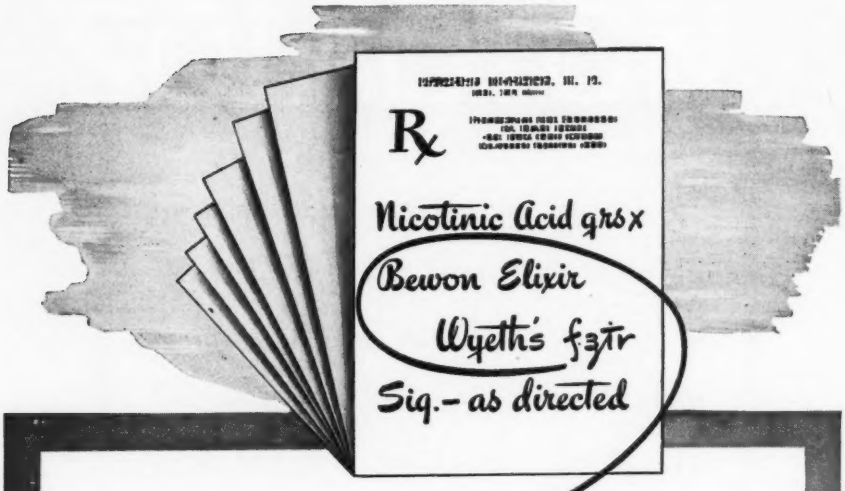

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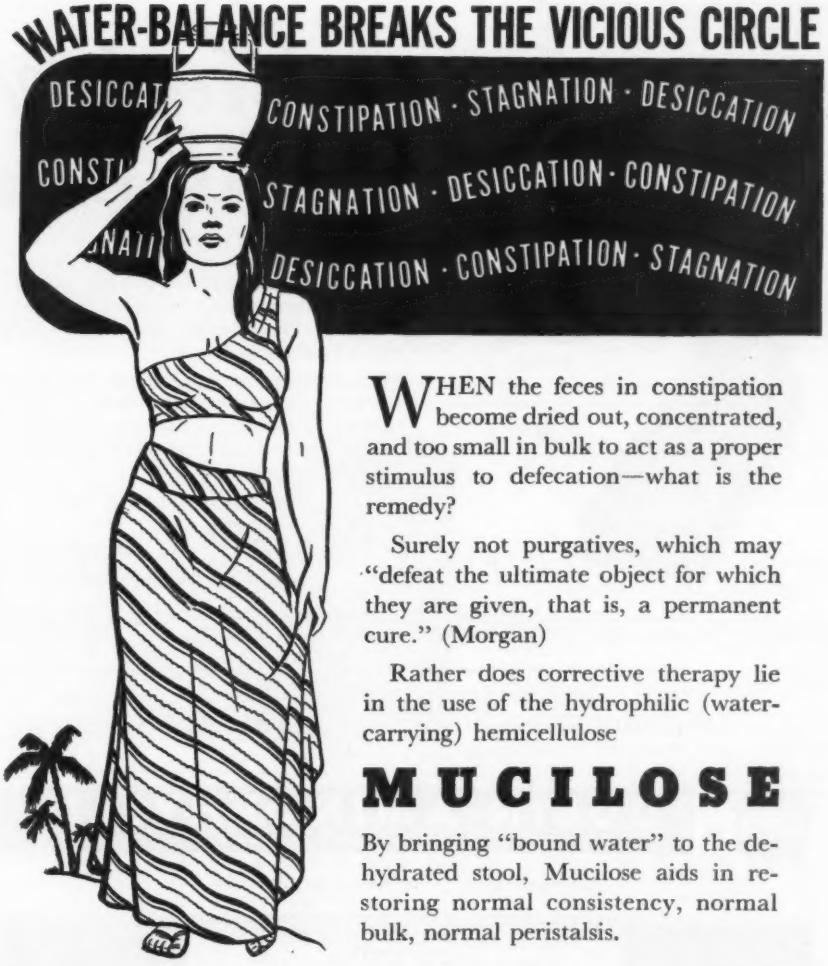

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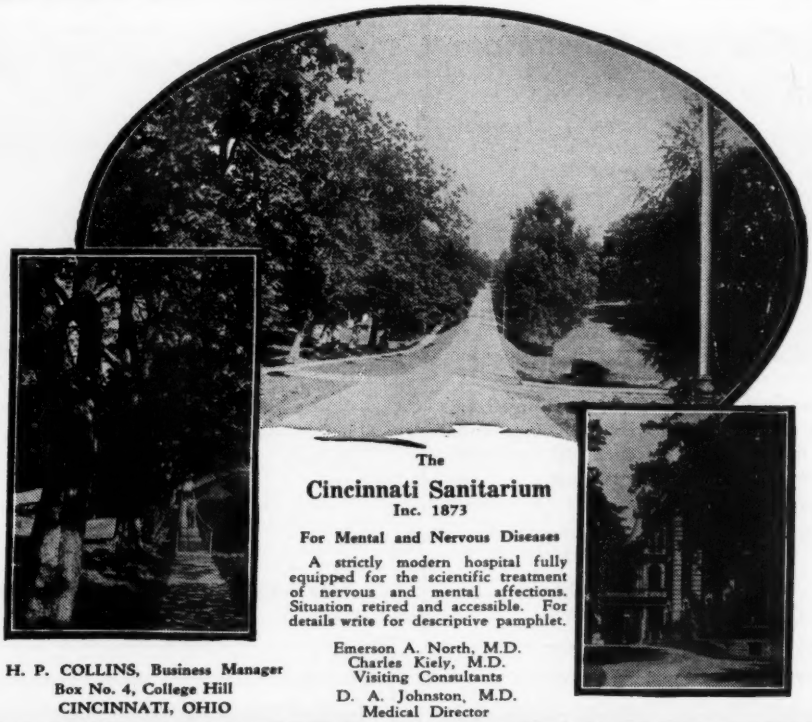

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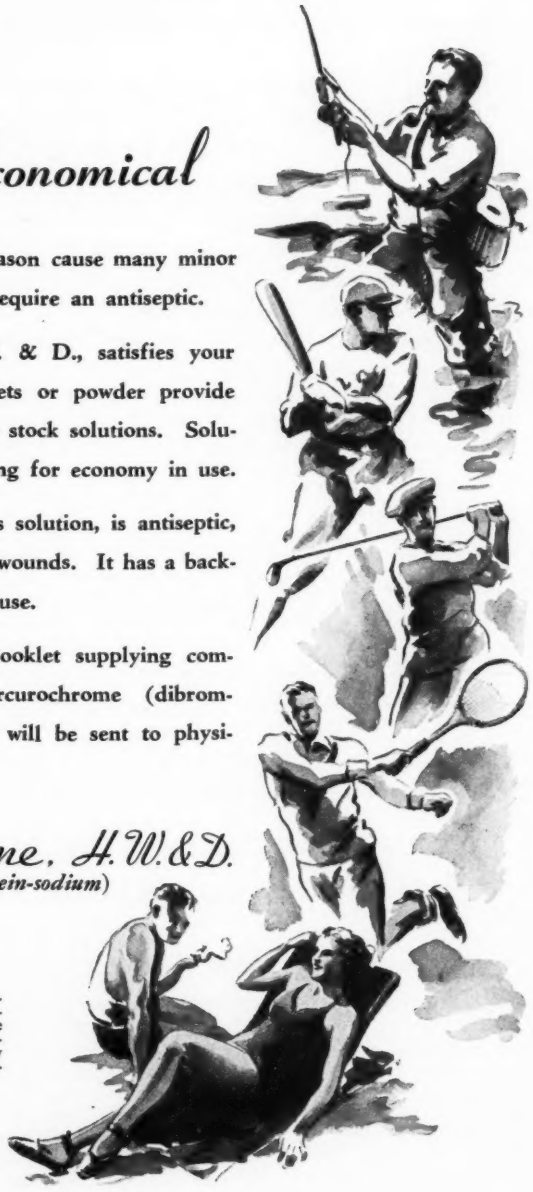

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Volume 34 OCTOBER 1941 Number 10

CONGENITAL PSEUDARTHROSIS OF TIBIA AND FIBULA*

REPORT OF FIFTEEN CASES

By J. HIRAM KITE, M.D.
Atlanta, Georgia

Congenital pseudarthrosis of the tibia and fibula is a false joint or fracture of the tibia and fibula in children, which seldom, if ever, heals spontaneously, and can be induced to unite only with great difficulty by bone grafting. The prolonged loss of use of the limb is a very perplexing problem. At the meeting of the British Orthopedic Association last year the president, speaking on bone grafts, said that "no satisfactory way of grafting birth fractures of the tibia has been found."

This condition has been described under several descriptive terms. A recent article[1] refers to these as "Birth Fractures" of the tibia and fibula. This is not a good term, as the trauma of being born has nothing to do with it. Occasionally the tibia is found to have a pseudarthrosis at birth, but this fracture behaves entirely differently from those fractures produced during the delivery. The normal tibia is seldom fractured during a delivery. It is the humerus and femur and clavicle which are fractured. These bones heal very rapidly, with or without treatment. You cannot keep them from healing, while you cannot make the pseudarthrosis heal. Of the fifteen cases studied, only one showed a fracture at the time of the delivery.

It is a "congenital" process, in that the children are born with an abnormality of the tibia, but this condition is not inherited (*Fig. 1*). The tibia before it has fractured shows an anterior and lateral bowing, with the lower half of the shaft smaller and more dense, and at times cystic. As Brailsford[2] might say, there is a defect in the architect's plans for this tibia, and also in the building materials themselves. Certain of the bone elements have entirely lost their power to reproduce bone. In fact, it seems that there is present a hyperactivity of the bone-destroying elements.

It has been thought that this condition is a form of osteitis fibrosa cystica. It is generally accepted now that osteitis fibrosa cystica is a general systemic disease, directly or indirectly connected with hyperparathyroidism, while congenital pseudarthrosis is a localized condition. The blood studies in congenital pseudarthrosis show no change in the calcium or phosphorus content. These children are normal otherwise. This condition has been called "congenital localized osteitis fibrosa cystica of the tibia, associated with pseudarthrosis."[3] The term "osteitis" suggests an infection, but the microscopic reports[4] show no round cell infiltration or other evidence of infection.

Many writers have thought that congenital pseudarthrosis is due to a congenital disturbance of the circulation of the bone. Others have thought that this might be the result of some involvement of the nerves to the bones. Gould[5] showed that von Recklinghausen's disease is more than a disease of peripheral nerves and skin, that a frequent symptom is softening of the bones similar to osteomalacia. Brooks and Lehman[6] called attention to a variety of changes which occur in bones in von Recklinghausen's neurofibromatosis, but did not report any pseudarthrosis.

Ducroquet,[7] Barber[8] and others have called attention to the fact that certain of these cases of pseudarthrosis are associated with von Recklinghausen's neurofibromatosis. Barber's four cases were all girls and all foreigners. All my cases are native Georgians. None of my cases presented the extensive involvement shown by

*Chairman's Address, Section on Bone and Joint Surgery, Southern Medical Association, Thirty-Fourth Annual Meeting, Louisville, Kentucky, November 12-15, 1940.

*From the Scottish Rite Hospital for Crippled Children, Decatur, Georgia.

these authors. Six did show "cafe-au-lait" spots and one of these a few neurofibroma molluscum. This association should be always kept in mind in taking the history and in examining these patients in the hope that it might shed some light on the etiology of the condition.

These tibiae do present pseudocyst-like areas, which are often unilocular, and which may enlarge, coalesce, or entirely disappear. Bones are not static, but are in a constant state of flux. This is true for the pseudarthrotic as well as for normal bones. X-rays made from time to time show almost constant changes in these vacuolated areas (*Figs. 2* and *3*). The cysts may appear in any part of the bone. When they are near the junction of the middle and lower third of the tibia, a fracture may occur following very slight trauma. It seems that the process which is destroying bone and producing the cyst continues to be active. Callus is prevented from forming, and the bone ends gradually become smaller in diameter and more dense. In every case in this series the distal end of the proximal fragment became more or less concave and the proximal end of the distal fragment became pointed.

This condition is typical both clinically and in the x-ray. An effort should be made to recognize it, as it may save embarrassment some day. If the bowing of this condition is mistaken for rickets and an operation is done to correct the deformity, the chances are that nonunion will follow. In three patients in this series this mistake was made elsewhere and all resulted in nonunion.

It has been stated that the fracture was at the junction of the middle and lower thirds of the tibia. The exact location of this fracture level has been questioned. In nine of my cases x-rays were available for study before any operative procedures had been done. In these, measurements were made of the length of the upper and lower fragments of both the tibia and

Fig. 1

All of the illustrations used in this paper are of the same patient. When the baby was three weeks old it was first noticed that the left lower leg was crooked. This deformity gradually increased.

X-ray appearance of the tibia and fibula at fourteen months. This cannot be called a congenital pseudarthrosis of the tibia at this time because the tibia has not fractured, but this is the typical appearance of the congenital changes seen in the tibia before the fracture occurs. The shaft of the tibia is smaller and sclerotic in the lower half, with cystic areas near the middle and above this point. A spontaneous fracture of the fibula has already occurred. There is the usual marked anterior and medial bowing which precedes the fracture of the tibia.

Fig. 2 Fig. 3

Fig. 2.—At fourteen months a brace was applied to protect the tibia from fracture. At sixteen months, while the child was playing on the bed without his brace, the tibia fractured. X-rays show the condition when the patient was next seen at thirty-three months. There is a pseudarthrosis of the tibia and fibula, with sclerosis of the fragments. The proximal end of the distal fragment is pointed. The proximal fragment shows a large cyst. The medullary canal near the end of each fragment has been sealed off by the sclerosis of the bone. There is no evidence of callus. Near the lower end of the distal fragment there has been a green stick fracture, which is not an uncommon finding in these cases.

Fig. 3.—Operation was refused and the patient was not seen again until the age of 6, when this x-ray was made. The green stick fracture has healed, and the cyst in the upper fragment has been partially obliterated by the deposit of bone. The bones show extreme atrophy and little increase in size during the three years.

Case	Age at Time X-Ray Was Made	Length of Upper Fragment of Tibia	Length of Lower Fragment of Tibia	Per Cent of Distance from Lower End	Length of Upper Fragment of Fibula	Length of Lower Fragment of Fibula	Per Cent of Distance from Lower End
4	9 mos.	6.2 cm.	4.0 cm.	39	7.3 cm.	2.5 cm.	26
5	6 yrs.	16.6 cm.	8.0 cm.	33	18.4 cm.	5.0	21
7	2½ yrs.	10.2 cm.	7.3 cm.	42	8.5 cm.	4.9 cm.	37
8 2nd fracture	3 yrs.	12.5 cm.	4.0 cm.	24	9.8 cm.	2.7 cm.	22
" 3rd fracture	5 yrs.	14.6 cm.	5.5 cm.	27	12.5 cm.	3.5 cm.	22
9	2½ yrs.	11.0 cm.	3.1 cm.	22	No fracture fibula		
11	8 yrs.	17.7 cm.	7.5 cm.	30	15.3 cm.	4.9 cm.	24
13	1½ yrs.	9.7 cm.	5.5 cm.	36	8.5 cm.	4.0 cm.	32
15	7 mos.	5.6 cm.	3.5 cm.	38	5.0 cm.	1.8 cm.	26
				Average 32.3			Average 26.3

Table 1

fibula in the lateral x-ray (*Table 1*). Measurements in the anterio-posterior views are inaccurate because of the anterior bowing of the tibia. The average of these show that the tibia fractures at the junction of the middle and lower thirds, being 32.3 per cent of the distance from the lower end. The fibula fractures at a lower level, at about the junction of the third and fourth fourths, or 26.3 per cent of the distance from the lower end.

Probably every type of bone grafting has been tried on these cases by many of the most skilful surgeons in all parts of the world. Henderson[9] reports five cases with three failures, Colonna[10] reports three patients with one failure, and Putti reports thirteen cases with eleven failures. Henderson first called attention to the fact that prognosis was better in older patients and recommended that operation be postponed until after puberty.

REVIEW OF CASES

Incidence.—Fifteen cases of congenital pseudarthrosis have occurred in eight thousand orthopedic patients under 14 years of age, giving an incidence of nearly 0.2 per cent.

Sex is equally distributed. There were seven boys and eight girls.

Right and left sides shared alike. In seven patients the pseudarthrosis was on the right and in seven on the left, and in one both tibiae were involved, though only the left was fractured.

Onset of Deformity.—The deformity was noticed in most cases shortly after birth. In two

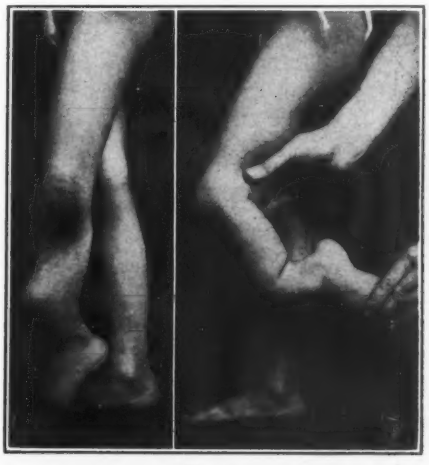

Fig. 4
Photograph at 6 shows the amount of free and painless motion at the point of the pseudarthrosis.

Time at which first fracture of tibia was noticed	Birth	1 Week	6 Weeks	2 Mos.	6 Mos.	1 Year	1½ Years	2 Years	2½ Years	6 Years	7 Years	8 Years
Number of children developing fractures at time indicated above	1	2	1	1	1	2	1	1	1	1	1	1

Table 2

of the cases no deformity was noticed before the fracture.

Time of Fracture.—In only one case was fracture noticed at time of delivery. More than half of the fractures occurred during the first year of life. Another quarter occurred before the children reached their second birthday, while three of the fractures did not occur until the children were 6, 7 and 8 years old (*Table 2*). Henderson divides these cases into three groups, and puts these older ones in a group to themselves. Gasne[11] divides them into four groups. Since they all present the same pathology and behave alike, no division will be made here.

One boy did not have his fracture until he was 6 years old. He had two unsuccessful operations on his leg to get union before admission, and it was only after our second bone graft that we obtained union. A girl was 8 when she had her fracture. She spent a year elsewhere in casts because of the nonunion, and then we did a bone graft which was successful. Twenty-one months later she refractured this same tibia and had another nonunion, which

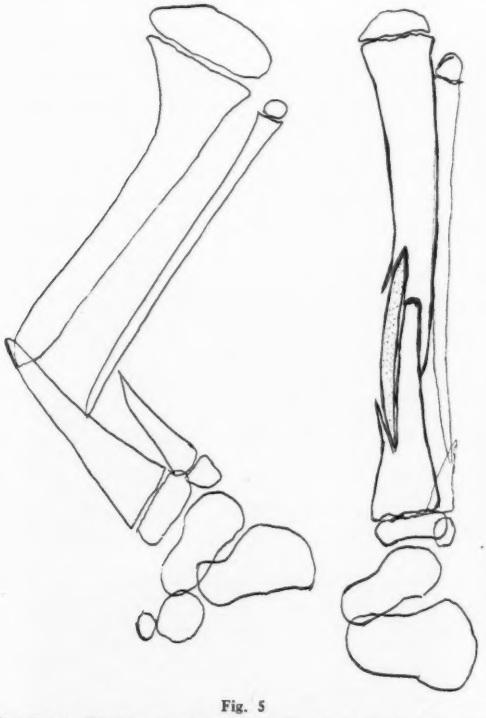

Fig. 5

Drawing to illustrate the "bone flap onlay" graft used. The lower fragment was mortised into the upper fragment. The medullary canal was opened. An onlay graft was obtained from the upper end of the same tibia, and was placed across the point of fracture. Each end was slipped under the bone flaps raised from each fragment, which gave firm contact and good immobilization.

Fig. 6 Fig. 8

Fig. 6.—Lateral x-ray made a few days after operation shows the close contact of the graft with the tibia. The alignment is good and the mortise can scarcely be seen.

Fig. 8.—X-ray to show the actual appearance of the tibia and graft a few days after operation.

required another bone graft for union. These two cases are mentioned to show that these older patients follow the pattern set by the younger patients, and that they might all be discussed together.

Nature of Tibia before Occurrence of Fracture.—We studied a number of pseudarthrotic x-rays after the fracture had occurred, wondering what the bones looked like before they had fractured. Finally we saw a patient before the tibia had fractured. We now have four cases.

The first was a fourteen months old boy with an anteriar and medial bowing of the tibia and with a fracture of the fibula (*Fig. 1*). The bones showed extreme atrophy. The upper half of the tibia showed a large cyst and the lower half was dense and sclerotic, and about one half the normal size.

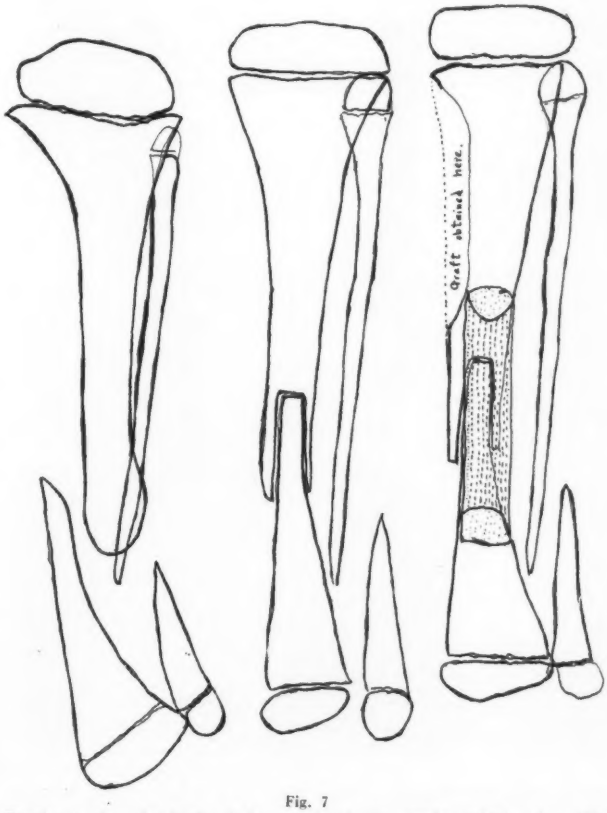

Fig. 7

Drawing to show the details of the operation in the anterior posterior view. The lower fragment was mortised into the upper fragment. The onlay graft cut from the upper end of the tibia was placed across the fracture line and the ends slipped under the bone flaps turned up from each fragment.

Mechanics Producing the Fractures.—It seems to me that the best explanation of this condition is that the atrophy and cysts cause softening and diminution of the diameter of the shaft of the tibia and allow it to bend. This throws more strain on the fibula, which is essentially normal, and does not bend readily. This constant abnormal stress on the fibula causes an absorption of bone salts and the fibula finally fractures. In nine of the cases there was a fracture of both bones when first seen. In the four mentioned above the fibula fractured first in three. One was protected by a brace and the tibia did not fracture. Two patients had fractures of the tibia without any fracture of the fibula. In one case in which the condition was bilateral, the opposite tibia and fibula were prevented from fracturing by the patient's wearing a brace. Once this tibia showed a fracture line, but this healed while the patient was walking on it wearing a brace, and it has not broken at the age of 12.

Results of Operation.—In counting the number of operations necessary to obtain union in these cases, I will not report those done before the patients were admitted, because too little information can be obtained. Some of the operations were done by general surgeons, but most of them were done by orthopedists. Four of these patients had a total of eleven operations before admission. *Case 2* had six operations, *Case 6* had two, *Case 9* had one, and *Case 11* had two bone graft operations, all with nonunion.

Several methods might be used in reporting results following operation. One method might show a better score for the operator, but another method might better reflect the true picture of the condition. One author reports 66 2/3 per cent success, in that he got union in two cases out of three. The third finally came to amputation. However, he did five bone graft operations upon these three patients, so his percentage of successful grafting should have been 40 per cent. This does not give a true picture of the condition, be-

cause these three patients had fifteen bone graft operations by this surgeon and previous surgeons, so there were only two successful operations in fifteen attempts, giving a 13 per cent success in bone grafting congenital pseudarthrosis.

Only thirteen of the fifteen patients in this series have been operated upon. The fourteenth patient has not fractured as yet. The parents of the fifteenth patient refused treatment when the nature of the condition was explained to them. The thirteen patients submitting to operation had twenty-eight operations, these being done by Dr. Michael Hoke, Dr. Lawson Thornton and me. Thirteen of these resulted in nonunions, fourteen in bony union, and one is too recent to report. Three of these patients (*Cases 5, 7* and *12*), after receiving a firm bony union, later refractured the same tibia and developed another pseudarthrosis. These refused to heal with good immobilization in casts for three months or longer, but firm union was obtained after a second bone graft. They are included in the fourteen unions.

Case 5 got solid union following a bone graft operation at the age of 7, and began walking

Fig. 9 Fig. 10

Fig. 9.—X-ray a year and a half after operation shows solid bony union with restoration of the medullary canal. A few radiolucent areas are still present. Casts were worn for fourteen months after operation, and the patient had been wearing a brace for four months when these x-rays were made.

Fig. 10.—Two years after operation, the patient fell at school and refractured the left lower leg. Casts were worn for two months. No callus was deposited and there was no evidence of union. These x-rays show the condition before the second operation.

with a brace in five months. Sixteen months after operation she fell off the bed when she was not wearing her brace and refractured the same tibia. *Case 7* broke his leg at school, while wearing a brace, two years after a successful bone graft operation, and developed another pseudarthrosis (*Figs. 9* and *10*). Another bone graft was done and union was obtained, and he has had no further accidents after five years. *Case 12* had her second fracture 21 months after her operation, and with no known trauma. She gradually developed pain and motion while wearing a brace. Casts, without and later with a walking iron, were tried, but x-rays continued to show more absorption at the point of pseudarthrosis, so a second bone graft was done, which was successful.

The age at which the grafting should be done comes in for careful consideration. Henderson was probably the first to recommend that the operation be postponed until after puberty. Colonna and others have reaffirmed this opinion. Most writers recommend that these children wear some type of brace until they have reached puberty. Even when this is done, the child is able to bear little weight on the fractured leg, and the leg wastes away and becomes very short. The bone fragments become pointed and develop more space between the fragments.

It is my belief that attempts should be made to graft these cases early in life, in the hope that union may be obtained, and that by weight-bearing the leg may develop more normally, and become more useful. The chief reason we get nonunion is that the local condition itself destroys bone instead of producing it. The bone does not produce callus spontaneously, and even after bone grafts have been applied very little callus can be seen. In fact, there is a very strong tendency for nature to absorb the graft, which it does completely in many cases.

The chief factor which seems to favor union is good internal and good external fixation. This is difficult to achieve in small children. In young children, the bones are hardly larger than a pencil and often are too small for the motor saw to be used. Even drilling a 7/64-inch hole for a vitallium screw destroys most of the cortex of the shaft at that point. Again, the sclerosed bone in the pointed ends is very brittle and cannot be split for the insertion of the graft without the risk of fracturing it. After the graft has been placed, it is difficult to apply a snug cast to the leg to get good external fixation in a small child.

To show the importance of good external fixation, one case (*Case 8*) was induced to heal by external fixation alone. This case showed a large cyst occupying the lower half of both the tibia and fibula. After ten months in casts he had firm union. He walked from the twelfth to the eighteenth month in a cast with a walking iron, and then a brace was applied, which was worn until he was 36 months old. He then got a new brace, which did not give as good support. While wearing this he developed another fracture. He again healed solidly after a year in casts with a walking iron, and without an operation. He then walked with a brace for another year before he developed another fracture, and this time he did not heal with casts, and had to have two bone graft operations before union was obtained.

The fourteen successful bone grafts were done at the following ages: 4, 5, 6 and again at 8½, 7 and again at 8½, 7, 7½, 8½, 10½, 11½, 12, 13½, and 14 years. The average age at which a successful graft was done was 8.8 years. It is quite true that the older the patient the greater the chance for a successful bone graft.

Factors influencing the taking of bone grafts were studied. Of the twenty-eight operations, a motor saw was used in only four. The remainder were done with a hammer and chisel. Three of the four done with the saw were successful, giving 75 per cent, which is much better than the 48 per cent obtained with a chisel. The successful grafts were done at the age of 4, 6½ and 8 years. The one failure was at the age of 2. However, with many other factors playing a part, such a small series must be taken with caution. The superiority of the motor saw may not be quite so great as these percentages indicate.

The percentage of takes has been slightly higher when the bone was taken from the opposite tibia. Many parents object to having anything done to the normal leg, and for this reason the graft has frequently been obtained from the same leg. Taking it from the same leg works better in older patients than it does in the younger ones. When the bone was taken from the same tibia we had five successes and six failures, giving 45 per cent success. When the bone was taken from the opposite tibia we had nine successes and seven failures, giving 56 per cent success.

The type of graft to be used is always an important question. Various grafts were tried in this series. None were used frequently enough

for the percentages to be of much value, yet expressing the results in percentages gives some indication as to their value (*Table 3*). Eight inlay grafts were used, four of which were successful. Three diamond-shaped inlays (Gallie[12]) were used, with only one success. The failure of these is probably due to the fact that it is difficult to split the pointed fragments to receive the diamond-shaped graft, without fracturing one side, and if one side breaks off there is little to hold the graft. Free shavings without a massive graft were used three times, with one success. A portion of the opposite fibula was used as a free graft once without success. One onlay was held in place only by closing the periosteum and muscles over it. This was unsuccessful. In two cases two or more thin shavings of bone were placed about the fragments as onlay grafts and these were held in place by tying silk ligatures tightly around the grafts and tibia. Both were failures. Vitallium screws were used in the last two cases. One was successful, and the second is too recent to report.

The type of graft used most often, which has given us the best results, has not been described before, so far as I know. This was used eight times with seven successes, giving 87.5 per cent good result.

This graft was devised by Dr. Lawson Thornton in 1934, but was never reported. It might be called a "sliding-end-under-boneflap-onlay" graft, or a "boneflap onlay" graft (*Figs 5* to *8*). The bone graft which is obtained with a chisel tapers down to a fishtail at each end. The two fragments of the tibia are notched together in any manner which may appeal to the operator, so as to place the shafts of the two fragments on the same level. The area to receive the graft is then made flat by removing a few bone shavings. The chisel is then inserted into the side of the shaft of each fragment where the graft is to be placed. A bone flap is raised as the chisel is driven obliquely into the cortex, going from the fracture toward the end of the bone. After these two boneflaps have been raised, the ends of the graft are slipped under each flap. The elasticity of these boneflaps holds the graft firmly in place. As a rule, no pins, screws or other form of fixation is used. This type of graft works as well in small bones as it does in large ones.

Going into a little more detail, I should say that before the graft is placed the sclerotic ends of the bones are opened so as to expose the medullary canal. I do not cut off the ends of the bones to open the canal, or to place them squarely end on, as has been recommended, because I believe we should preserve all bone possible in this region. I open the medullary canal by drilling a row of small holes in the anterior cortex and cutting the bone out between these holes with a thin osteotome. In this way the fragments are not fractured. Often they can be mortised together to give additional support. After the graft has been placed, curets full of medullary bone from the donor area are packed about the bone ends and the graft. Any additional small bone shavings available are packed about the point of the fracture. The periosteum can be closed over the ends of the graft, but along the middle only muscles can be brought over the bone. The graft is usually

Type of Graft Used	No. of Cases	Union	Nonunion	Percentage Successful
Inlay	8	4	4	50
Diamond inlay	3	1	2	33
Fibula	1	0	1	0
Bone shavings	3	1	2	33
Onlay held by suture of soft parts over it	1	0	1	0
Two or more long, thin onlay grafts held by silk over grafts and around tibia	2	0	2	0
Vitallium screws in onlay grafts	2	1	One too recent to give result	
"Boneflap" onlay	8	7	1	87.5

Table 3

placed on the side of the tibia next to the fibula, so that it is buried deep beneath the muscles, which gives it a better blood supply (*Figs. 11* and *12*).

All deformity must be carefully corrected before the graft is inserted. If this is not done weight-bearing will produce a stress on the bone which may favor resorption of the grafted area.

External fixation is almost as important as good internal fixation. I have seen casts which extended only to the upper thigh in a small child become loose after a few days and slip, and probably be the cause of failure. Sometimes I use a previously-prepared and padded sterile wooden splint to immobilize the leg. After the bones have been prepared to receive the graft, the foot and lower leg up to the lower end of the wound is bandaged to the posterior splint, and the leg from the upper end of the incision to above the knee is bandaged to the splint. This gives a very good fixation of the leg. Then the graft is inserted. In this way I am sure that the graft is not loosened during the closure of the wound and the application of the cast, which is applied over the splint. I have used this seven times with five successes.

In small children the cast is continued up on the body as a spica so as to prevent torsion on the graft, which may occur when a straight leg cast is applied extending only to the upper thigh. If the wooden splint is not used, torsion strain is prevented by flexing the knee and hip. The stitches are removed through the smallest opening possible and the cast is not changed for eight or ten weeks.

A study was made to see what effect infection might have on the taking of the graft. It is very rarely that we have an infection in any of our bone operations. However, some of these legs have been operated upon previously, and the scars suggested that they had healed by second intention. In no case was there an infection of the wound where the graft was obtained. Seven cases showed more or less infection of the wound over the graft. In these cases the incision had been made through scar tissue and the skin was under tension where it covered a massive graft. Of the twenty cases which healed *per primam*, there were nine successes and eleven failures, or 45 per cent success. Of the seven which showed some infection, five were successes and two failures, giving 71 per cent union. Therefore, infection does not seem to interfere with a take. In fact a little infection seems to stimulate the production of callus, but I am not recommending this as a form of treatment.

If union is going to take place it will be present when the first cast is changed at the end of six to ten weeks. Those not showing union have all been immobilized longer, hoping for union. *Case 6*, which developed nonunion at the upper end of the graft, did unite after eight months in casts. The patient still has good union twelve years after the bone graft operation.

The patients were kept flat in bed without

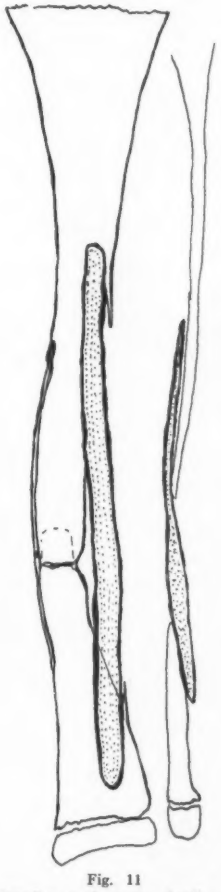

Fig. 11

Drawing to show the details of the second operation. A massive onlay graft was obtained from the opposite tibia. Each end was slipped under a bone flap as before. A long graft was used to give better fixation. Additional shaving grafts were placed about the fracture and about the fragments of the fibula.

weight-bearing for about three months and then let up with a cast and walking iron. The average time in casts for the entire series was 9.7 months. A snug fitting brace was then applied.

A word about braces is in order. The usual type brace attached to a shoe, and lacing up over the anterior surface of the tibia, does not give sufficient support. When a brace is desired, we make a plaster model of the leg. The two bars on each side of the brace touch the leg lightly on each side. These are attached to a metal foot piece, something like an arch support. There is a solid leather cuff covering the anterior surface of the leg. This is shrunk over the model and fits very accurately. This gives uniform pressure over the anterior surface of the tibia, which usually becomes bowed anteriorly a little in spite of our care. The leather cuff of the brace is laced in the back, over the calf (*Fig. 15*). The shoe can now be slipped on over the foot and the foot support, without putting any strain on the grafted area. Our earlier braces had a joint at the knee, but fractures have occurred when there was motion at the knee, so I am now using braces, especially for small children with no joint at the knee. The brace extends to the upper thigh. It is diffi-

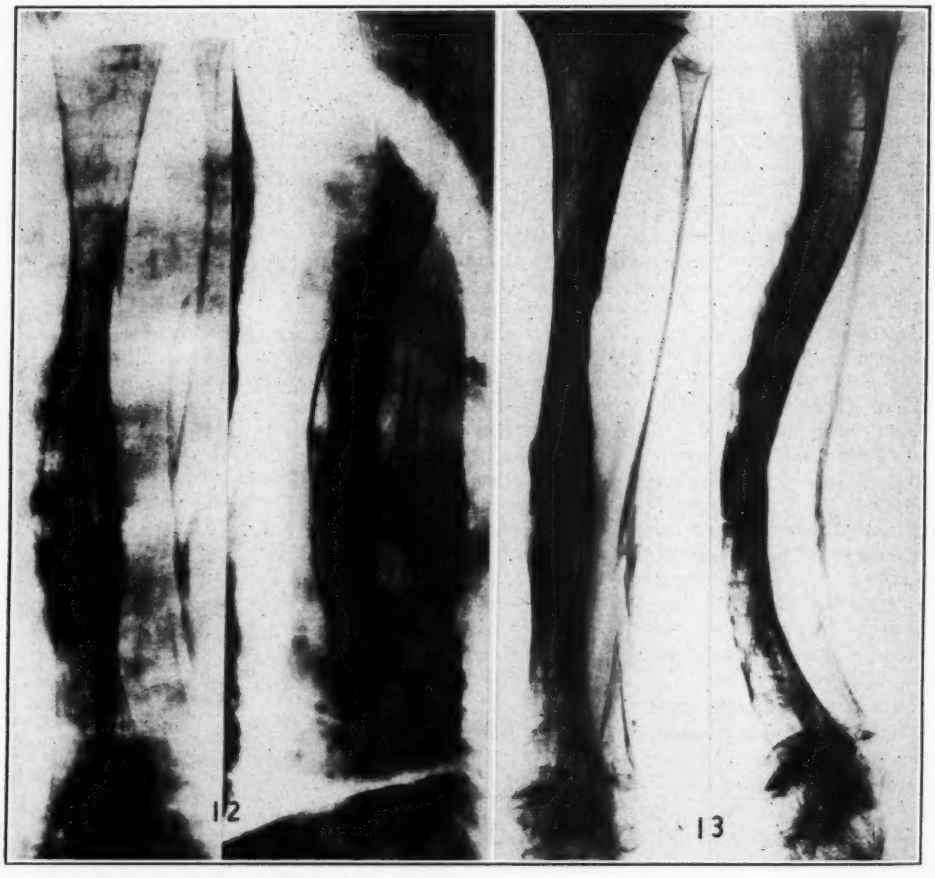

Fig. 12 Fig. 13

Fig. 12.—X-ray to show the graft a few days after the operation. The graft was 12 cm. in length.

Fig. 13.—X-ray six months after operation shows solid union at the point of fracture, with the graft intact, but somewhat smaller than it was six months before. The grafts about the fibula united to the proximal fragment, but did not unite with the distal fragment.

Fig. 14 Fig. 15

Fig. 14.—Photograph of the leg a year and a half after the last operation shows that the left leg is straight. The left leg is 6 cm. shorter than the right.

Fig. 15.—The brace used gives better support than the average brace. The lateral bars fit close to the leg. These are attached to a foot plate. The leather over the anterior surface of the lower leg was shrunk over a plaster model of the leg to give an accurate fit. The brace laces in the back. This brace gives good protection to the leg and prevents any strain on the fracture, even in putting on the shoe. A cork lift is used to compensate for the shortening. The patient has good union now, five years after the last operation.

cult to know when to leave the braces off in these children, as the bone in the x-ray is atrophic and friable in appearance. Many of these cases are still wearing a brace for support.

All of these children have a great deal of shortening by the time they are grown, so that the leg is never so good as might be desired, even though firm union has been obtained. The earlier the union is obtained, the less the shortening. The average shortening for six of these cases who have reached the age of 16 is 10 cm. We use a cork lift, or a U-shaped extension, until the child has reached his full growth, and then have an artificial foot made. This is made so that the patient bears his weight on his foot, which is pitched down in as much equinus as possible. He stands on top of the artificial extension and walks better than he might if he had only a stump.

It has required a great deal of hospital time for these patients. In fact, so much time that we are tempted to wonder sometimes if it would not be better to do an amputation when they are first seen and not try to graft them. They usually have to wear some sort of extension in the end. The average time of the twelve whom we hope we have finished was 393 days each in the hospital. At $3.00 a day, this amounts to nearly $1,200.00 for each patient. To this should be added $75.00 to $150.00 for braces during the years under treatment. An amputation would be the most economical treatment, but is not the best orthopedic surgery. I believe that a living foot below par is better than a wooden foot, which many of these patients are not financially able to replace when a new one is needed.

Of the thirteen cases operated upon, one of the earlier patients in the series left with a nonunion and did not return. One is too recent to be sure of the result. This gives eleven cases with good union at this time, which justifies the continuation of bone grafting operations.

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FIBRIN CALCULI*

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Under the title fibrin calculi, we propose to discuss those soft putty-like masses which at times form in the renal pelvis. These may occur either as a primary calculus or develop following the removal of another type of stone. It is important not to confuse fibrin with phosphatic calculi. The latter are produced by urea-splitting bacteria. Later on, the mechanics of the formation of these two different types of stone will be considered. Fibrin calculi always lie free in the renal pelvis and apparently never extend into the minor calices. They are thickest where pelvic tension is least. The symptoms of fibrin calculi are the same as those produced by other types.

The physical characteristics of fibrin stones are distinctive. These masses are soft and rubber-like in consistency and can be molded to moderate extent without breaking. The usual color varies from a pale to a bright yellow; but, at times, blood pigment may give them either a red or a dark brown color. The inorganic content is not constant in any two fibrin stones nor in all portions of the same calculus. Some portions will feel gritty, while other areas are smooth and no roughness can be detected by palpation. On exposure to air these masses quickly dry; after which, they easily break into fine fragments. When fixed in 10 per cent formalin, they shrink, become firm like tissue, and can be easily cut into sections, stained, mounted, and studied under the microscope. Under this latter only, can be seen cellular debris lying amid large numbers of fibrin strands (*Figs. 1* and *2*) with here and there a vacancy formerly occupied by phosphatic crystals.

The fibrin of these stones can be digested by the use of suitable enzymes. Such digestion leaves behind the inorganic material, which, having no cohesion, quickly falls apart. Caroid supplies such an enzyme. In several of our cases, lavage of the renal pelvis with a 1 per cent caroid solution digested the fibrin, after which the patient was able to pass the smaller phosphatic debris.

Fibrin calculi have no uniform size. Frequently, the mass fills the entire pelvis. In other cases, many small masses about 1/4 to 3/16 of an inch in diameter continue to form. These being small, pass down the ureter, causing varying degrees of colic. The patient may void three or four of these pellets at a time. One individual had a penny match-box full of these small fibrin stones.

The larger masses are moulded to the general contour of the pelvis, but I have never seen them extend into an infundibulum. When the inorganic content is low, no shadow can be seen in the plain film. On the retrograde pyelogram, they register as a negative filling defect suggestive of tumor or blood clot (*Fig. 3*).

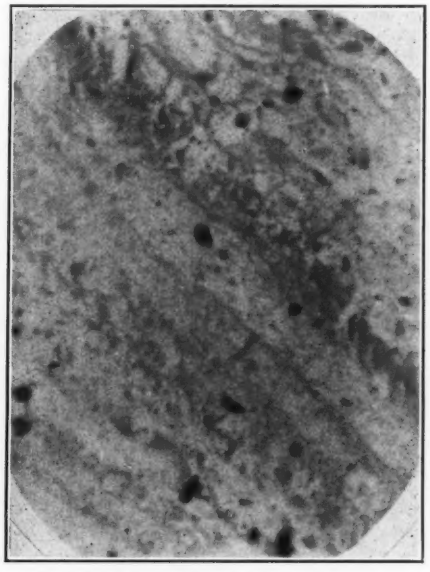

Fig. 1
Low power photomicrograph of a fibrin stone; note fibrin network.

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Macroscopic examination of the removed masses suggests coagulated plasma. A mass having similar qualities may be obtained by soaking cutaneous crusts or scabs in water. These fibrin masses can be moulded with the fingers in much the same way. Having once felt a fibrin mass, future identification is easy.

Inspection of the opened renal pelvis, which has contained one of these stones, reveals a red swollen edematous mucous membrane with scattered areas of petechial hemorrhage. Microscopic sections of the pelvis wall indicate an acute inflammation with swollen epithelial cells and leukocytic infiltration. In many areas, the epithelium has been completely denuded and the muscular layer exposed (*Figs. 4, 5, 6, 7*). A similar lesion on a cutaneous surface would pour out quantities of lymph with the formation of large crusts. There is no valid reason for not assuming that this lesion of the renal pelvis does pour out lymph which, coagulating in the urine, forms a fibrin mass. Varying amounts of urinary salts are deposited on its surface as it grows.

Bacteriologic study has failed to prove any one organism the etiologic factor, although infection has invariably been present. Gram-positive cocci and gram-negative bacilli have been found. Stained smears of the material usually show the same types of bacteria as are found on culture: staphylococci, streptococci, or

Fig. 3
Negative filling defect of proven fibrin stone in renal pelvis.

Fig. 2
High power dry photomicrograph of fibrin stone.

Fig. 4
Section of pelvic wall. Low power photomicrograph showing denuded mucosa membrane. This pelvis contained a fibrin stone.

Fig. 5

High power dry photomicrograph showing denuded mucous membrane and exposed blood vessels.

gram-negative bacilli, or a mixture of these organisms. Rarely, the organisms possess the power to split ureas; and then, but feebly.

Fibrin stones are comparatively rare. The few cases reported in the past are probably due to the lack of distinction between true phosphatic calculi and these. Phosphatic calculi are hard and gritty and cannot be molded; indeed, any attempt at molding will fracture the calculus. Urea splitting organisms have been present in those instances of phosphatic calculi studied.

The textbooks rarely mention fibrin calculi and then only very briefly. No discussion of etiology has been found in any of the reference works. Due to the many evident difficulties inherent in the problem, scientific solution cannot be expected at present. Many suggestive facts are available upon which to construct an hypothesis. Bodansky and Bodansky quote Schade as stating that fibrin stones belong to Type I stone or "pure colloid concretion." These Schade conceived as "produced by the irreversible precipitation of some hydrophile colloid." All this may be true, but it would appear that the denuded pelvic mucosa would certainly pour out large quantities of plasma; and on occasions blood, which, coagulating in the pelvis,

Fig. 6

Low power photomicrograph of wall of a pelvis which contained a fibrin stone. Pelvic wall looped about so that denuded mucous membrane can be seen.

Fig. 7

High power dry photomicrograph of *Fig. 6* showing absent mucous membrane and infiltration of pelvic wall with leukocytes.

produce the fibrin mass. The whole process would be "irreversible." Microscopic sections of the pelvic wall (as mentioned above) give visual proof of such denudation.

In one of the cases with bilateral fibrin stones or masses, in which nephrostomy tubes had been inserted following the surgical removal of the stones, observations were made at several periods on the urinary pH of each kidney. One kidney would secrete urine with a pH of 5.5 while the other kidney would secrete urine with a pH of 8. Without any change in the medication or variation in any physical factor affecting the patient, the kidney which had been secreting urine with a pH of 5.5 would secrete urine with a pH of 8 and the kidney secreting urine with a pH of 8 would secrete urine with a pH of 5.5. This was so astounding that careful checking was done to verify this switching of pH.

Dr. Nash and his co-workers have demonstrated that the kidney itself has the power to manufacture ammonia. So far, nothing further has been done with this fact. We have no knowledge of what stimulates or inhibits this peculiar function. In the case cited above, the excretion of urines of different pH is unquestionably dependent upon this property of ammonia manufacture; it is self-evident that the same materials with the same pH were supplied to each kidney.

The infection present in the wall of the renal pelvis, as evidenced by the microscopic sections studied plus the more or less constant alkaline reaction of the urine, probably plays an important role in the pouring out of serum onto the pelvic mucosa. It was noted that, with the cessation in the formation of fibrin calculi, the urine pH decreased to normal, that is, 6.0 to 6.5.

SUMMARY

(1) A definite type of renal stone or mass has been described.

(2) The lesion of the pelvis wall accompanying this type of stone has been demonstrated.

(3) A theory covering the possible mechanism of production of these calculi has been submitted.

(4) The above conclusions have been based on the study of ten cases of fibrin calculi.

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DISCUSSION (*Abstract*)

Dr. M. K. O'Heeron, Houston, Tex.—That fibrin calculi are rare is evidenced by the fact that in 1930 Pedroso, in a review of the literature, found only 25 cases reported since the condition was first described by Marcet in 1818.

Under the term, fibrin calculi, have been included those putty-like masses which form in the renal pelvis. This includes, in addition to the true fibrin calculi, the bacterial, the urostealiths, and oleostealiths.

Israel has divided fibrin calculi into four groups, which are:

(1) Masses of inspissated pus. They become very hard, show concentric lamination, and are frequently impregnated with salts of triple phosphate.

(2) Non-laminated bodies of a rubber-like consistency composed of fibrin.

(3) Large, black, non-laminated bodies that may fill an entire pyonephrotic cavity. These have the consistency of rubber, and are composed of an albuminous substance that Israel thinks is derived from blood.

(4) Small oval or facetted bodies composed of an albuminous substance and showing distinct concentric laminations.

In the 10 cases included in Dr. Allen's study, the calculi were confined to the renal pelvis proper, but other authors, including Jolly, Pedroso and Ogier Ward, have reported cases in which calculi were found in the calices. The pelvis and calices in these latter cases were dilated, and in Ogier Ward's case the calculi were bacterial rather than fibrin.

Dr. Allen has discussed the use of the enzyme caroid in the treatment of these calculi. Caroid along with other enzymes has become increasingly popular as a digestant of necrotic and fibrinous deposits such as are found in incrusted cystitis, badly infected wounds, and now fibrin calculi. When these calculi are diagnosed this method of treatment may save the patient an operation in many instances. However, in the past, the diagnosis of fibrin calculi has seldom been made before operation. The symptoms may be the same as those in any case of calculous disease, but the x-ray will show little if any calcification in the area of the calculus. Most authors stress the pyelographic findings which consist of a diffuse mottling or negative shadows, or both. As one would expect, hydronephrosis is usually present.

The discussion of the cause of these calculi and especially the description and photographs of the pelvis from which one has been removed are especially outstanding. The changes in the renal pelvis have been fully described: the edematous mucosa with the petechial hemorrhages, and later the erosion through the mucosa and the lymphocytic infiltration of the muscularis. These are the changes that one observes in any case of calculous disease, and are frequently considered to be the result of associated infection. That such lesions extrude lymph and tissue juices is a fact, beyond question, and it is now established that the common

kidney stone contains a colloid matrix which was probably derived from an ulcerative lesion in the pelvis or a calyx. From these observations one might well ask, is a fibrin calculus an incomplete form of the common urinary calculus?

Rosving has described a type of fibrin calculus which, in his opinion, was derived from a phosphatic calculus. He says that the stone formed in an alkaline urine. Later the type of infection changed, the urine became acid, and the phosphatic content dissolved, leaving behind the organic stroma.

Occasionally blood clots are retained in the urinary tract and become incrusted, forming calculi. Jolly has an illustration of one found in a bladder.

Pedroso's case occurred in a tuberculous kidney. Tubercle bacilli were demonstrated in urine from the involved kidney before the operation. Seventy small calculi were removed. They were laminated and contained mucin, albumin, fibrin, hemoglobin, calcium oxalate, and cholesterin. The medullar portion contained many bacteria and a few tubercle bacilli were seen.

Schmoral has said that they form about a bacterial nucleus and gradually increase in size by growth in the periphery.

Lauda has said that they can be formed by blood clots, necrotic filaments, or particles of pus. These substances are partly absorbed by bacteria and then form the albuminous framework of the calculi.

Schole explained the concentric stratification as being due to the tendency of certain albumins, especially fibrin, to form stratified layers.

Becker has stressed the importance of inflammation in their formation, and Ikoma succeeded in producing soft concretions *in vitro* with the products of bacterial metabolism in a fibrin medium.

Peiper reported a case of bilateral renal fibrin calculi composed of albumin, impregnated with uric acid. The nucleus was also composed of uric acid.

In fibrin calculi, as in other forms of urinary calculi, inadequate drainage must be given a foremost place in considering the cause. Several authors have reported cases of fibrin calculi in the bladder. Included in this group are Ikoma, von Lichtenberg and Blatt. Hyman has reported three cases that developed fibrin calculi in the bladder following suprapubic prostatectomy. All the cases so far reported have been associated with infected urine. The relationship between urinary tract obstruction and urinary infections has long been accepted.

Dr. Hubert K. Turley, Memphis, Tenn.—One of the cases referred to by Dr. Allen was the first I have seen of this condition. This patient was seven months pregnant and presented the usual symptoms of pyelonephritis of pregnancy. The usual treatment by indwelling catheters brought about a recession of her symptoms.

Within ten days there was a return of the pyelonephritis. This time there was no response to the treatment by drainage with indwelling catheters. In fact, it was very difficult to keep the catheter open. After seven days of no improvement the decision to empty the uterus was made, following which there was improvement and symptoms subsided for ten days.

Again there was a return of fever, rigors, pain and tenderness over the right kidney. Treatment by indwelling catheters was again tried with little recession of symptoms. After four days a decision to remove the kidney was made as pyelograms showed a marked destruction of the kidney by hydronephrosis.

On opening the kidney after nephrectomy we were surprised to find the pelvis filled with purulent material and a soft homogeneous mass about two inches square when spread out. This had the appearance of an old decayed cloth. Cultures made showed a streptococcus present. The patient's convalescence was uneventful following the nephrectomy.

Dr. Hamilton W. McKay, Charlotte, N. C.—One of the greatest problems we have to face in urological surgery in the Piedmont section of North Carolina is stone: its formation, proper management, proper surgery, prevention, and the great question of recurrence of stone after operation.

Drs. Gage and Beal have suggested a classification, from the standpoint of the consistency of stone, which is easy to remember and extremely practical. Therefore, we might consider three forms of stones in considering the subject as a whole.

(1) Hard uric acid and oxalate of lime calculi, which offer great resistance to crushing.

(2) The soft type composed of alkaline phosphates.

(3) The type of stone under discussion by Dr. Allen, namely: a softer substance than the alkaline phosphatic stone, different in appearance and consistency and very confusing both from a diagnostic standpoint and at the time of operation.

These putty-like masses spoken of as "blood calculi," "fibrinous concretions" and "colloidal stones" are not very common, are difficult to diagnose, and usually lie free in the kidney pelvis. They differ markedly from the other two classifications of the hard and soft stones in that they apparently never form in the minor calices. I, personally, have encountered only one or two cases of this type of renal calculi, years ago, and as I remember they presented very difficult diagnostic problems. X-ray examination shows only faintly outlined masses and it is difficult to say whether or not they are renal stones without extensive urologic proof.

Fibrin calculi represent one of our best examples of colloidal influence in the formation of kidney stone.

The two important clinical characteristics in this group of cases, as has been pointed out forcibly by the author, are the presence of hematuria, accompanied by kidney colic, and the soft, putty-like plastic mass, usually found free in the kidney pelvis, which is difficult to diagnose as compared with the other two classifications of renal stones.

Incidentally, Schade artificially produced calculi *in vitro* by simultaneously precipitating fibrogen to fibrin and calcium chloride to calcium phosphate in the same solution.

Dr. Allen (closing).—It is true that the preoperative diagnosis rarely is fibrin stone. I think the inherent difficulty of the situation is in making the preoperative diagnosis and securing a suitable explanation for what actually causes the formation of these stones. An unusual history is as follows:

The patient, acutely ill, presented himself with an obstructing uric acid calculus in the middle part of the right ureter. Relief was given by the introduction of

a catheter beyond the calculus. An intravenous urogram revealed a normal kidney on the left side. No catheterization of the left kidney was done. The obstructing calculus was removed from the right ureter surgically. The convalescence was uneventful, and yet, two weeks postoperatively the patient developed a fibrin calculus in his left renal pelvis. It is difficult to explain the formation of this calculus in this area by any theory available.

It may be that fibrin calculi are frequently the explanation for a considerable portion of recurrent stones. Evidently, geographical location plays a part in the formation of fibrin calculus; Dr. Kretchmer, with whom I discussed this type of calculus, said that he had not seen one in his practice.

It so happens that in operating at the Veterans' Hospital in Memphis I have treated patients from quite a number of states, and this is probably an explanation for a large per cent of cases in this series. They usually occur in groups of three or four. My first was seen in 1935 and the last one during the past year.

TRACHOMA PROBLEMS IN ARKANSAS*

By K. W. Cosgrove, M.D.†
Little Rock, Arkansas

Trachoma is a communicable disease; therefore, its control is the responsibility of the board of health. The disease is transmitted by direct contact and by customs such as the use of the common towel, wash basin, and crowded sleeping facilities. The infectiousness of trachoma has been conclusively proven by Thygeson.[1] The causative organism is considered to be a filterable virus. Unfortunately, neither microscopic nor serologic proof of the diagnosis can be obtained in most cases. The diagnosis must, therefore, be made from clinical findings only. For this reason it is important that the diagnosis be standardized, or an erroneous impression of the prevalence of the disease or of the benefits of any treatment would result. It is proper, then, to start this paper with a review of the diagnostic criteria.

Trachoma has been divided into four major stages with some subdivisions (MacCallan[2]). The first stage, known as the follicular, must be differentiated from follicular conjunctivitis and folliculosis. The presence of pannus (the infiltration of the cornea with blood vessels) is diagnostic of trachoma; a definite diagnosis of trachoma cannot be made without this sign, unless a proven case of trachoma has been seen in the same family. In folliculosis, the follicles are larger and more prevalent on the lower lid, while in trachoma the upper lid is more involved.

The second stage of trachoma must be differentiated from pneumococci, gonococci, swimming pool conjunctivitis and inclusion blennorrhea. In trachoma the under surface of the upper lid is red, velvety, moist, bleeds easily and has a purulent discharge. There are no blood vessels showing through, contrasting to the red conjunctiva streaked with blood vessels seen in the other conditions. The presence of pannus is diagnostic. A pannus is sometimes simulated by circumcorneal injection in an individual who has a marked level to the corneoscleral junction. This appearance will usually be present all around the cornea, but with the aid of a slit-lamp, no blood vessels will be seen penetrating into the cornea proper.

The third stage is similar to the second stage except for white or yellow scars seen through the redness of the upper lid conjunctiva.

The fourth stage is that of old, healed cicatrix, which gives an appearance unlike any other conditions. Pterygium, entropions and ectropions are common sequelae of the later stages.

The so-called "trachoma belt," as described by Rice,[3] extends across the central portion of the United States from the Allegheny Mountains to the eastern borders of Kansas and Oklahoma. It takes in most of eastern Kentucky and part of adjoining West Virginia, eastern and central Tennessee, a portion of southern Illinois, all the southern half of Missouri, and the northern half of Arkansas and the northeast corner of Oklahoma. The trachoma found in Missouri and Arkansas today originated from individuals with trachoma who came into these states from Tennessee and Kentucky in the early days of pioneer development.

The disease is very prevalent in the northern part of Arkansas (see *Fig. 1*). It is usually contracted in early life. The onset in 69 per cent of our series began before the patient was 20 years of age. The victim, by the time he might be so inclined, is in no condition to wander very far from his home district, and often his parents are blind and need his attention. These seem to be good reasons for the limited geographical location of the disease.

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†Supervising Ophthalmologist, Arkansas State Public Welfare Consulting Ophthalmologist, Arkansas State Board of Health; Associate Professor of Ophthalmology, University of Arkansas Medical School.

The present extensive campaign against trachoma in Arkansas was started in the last week of August, 1939, following a conference with Dr. Rice, United States Public Health Service. It was carried on by the Department of Public Welfare with the assistance of the State Board of Health. The first necessity was education, not only of the public, but of the profession, in the existing need of such a program. This was accomplished by the press, letters to schools and county officials, speeches to civic clubs, and in one district, a poster contest in the schools. The initial plan was for diagnostic and ambulatory treatment clinics in five districts covering the 30,000 square miles in the northern part of the State. It was indicated by records of those receiving blind assistance that trachoma was most prevalent in this section of the State. These clinics were started in the northeast and continued across the State at intervals. In each district an initial clinic was held after extensive publicity. A check-up clinic was held four or five weeks later before a new district was opened. To standardize the diagnosis, the supervising ophthalmologist was present at the first two clinics in each district. The subsequent twice-monthly clinics were conducted by local ophthalmologists. A full-time nurse arranged and managed all of the clinics. Two additional smaller districts were opened following requests from local physicians in the central part of the state. It is interesting to note from the standpoint of the spread of trachoma that in these districts one or two families, which had moved from the "trachoma belt," were found to be the center of an ever-widening circle of infected contacts.

Following further conferences with Dr. Rice, the management of the program was taken over by the State Board of Health on July 1, 1940. The present plan is two-fold: first, the continuation of the original clinics. These are conducted twice monthly by the part-time ophthalmologists, assisted, when necessary, by the consulting ophthalmologist. One full-time nurse attends all these clinics. The second portion of the plan is intended to overcome three of the suggested shortcomings of the original plan: first, the lack of close supervision of the patients during the period of extensive treatment; second, the failure of patients to return for re-examination after treatment; third, the difficulty of thorough case finding.

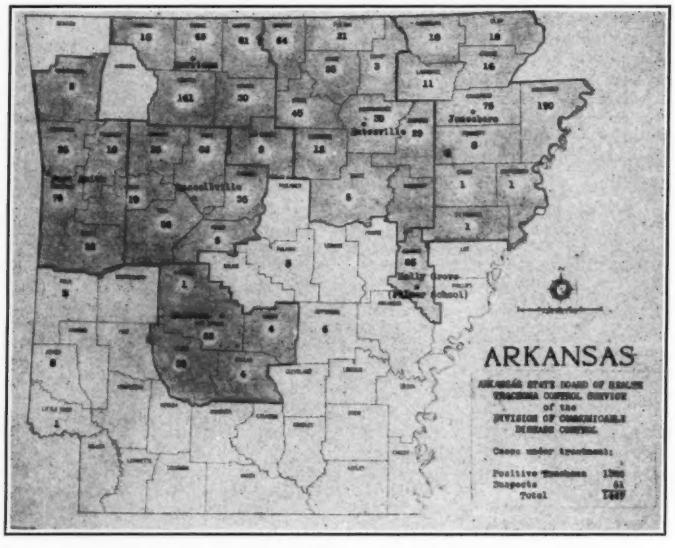

Fig. 1
Distribution of trachoma in Arkansas.

The second part of the program is carried on by a full-time physician and a nurse in one or more counties at a time. Their work includes seeking out of family, school and neighborhood contacts, in addition to the usual publicity. Fol-

lowing this case finding period, a clinic is held where cases are diagnosed and placed under treatment by the consulting ophthalmologist.

During the next four or five weeks the patients are met at weekly intervals near their home, progress is noted, hemoglobins are checked, and treatment continued by the full-time clinician. During this period many contacts are examined and any new cases seen are placed under treatment. After the period of extensive treatment, the clinician and nurse move on to another county, leaving the follow-up to the local health unit director and the district ophthalmologist.

During the thirteen months this program has been in effect 8,947 persons have been examined in the clinics. There have been at least an equal number checked by health directors and nurses before referring them to a clinic. One thousand three hundred and eighty-six of these were definitely diagnosed as trachoma and sixty-one suspected cases were placed under treatment. Of the positive trachoma cases, 246 have been rechecked at least three times, and several of these have had as many as twelve examinations: 237 have been rechecked twice and 352 have had only one re-examination (*Table 1*). There were 123 cases of fourth-stage healed trachoma which it was not considered necessary to re-examine. Two hundred and thirty-eight cases have not been re-examined because their county has had no scheduled clinic since their first treatment. One hundred and ninety-two cases, or 13.9 per cent, have not been contacted since the first examination. The disease was almost equally prevalent in each sex, 59 per cent being males. Three hundred and seventy-two, or 27 per cent, had vision of 20/200 or less, which is considered economically blind. There were 381 diagnosed as first stage, 343 second stage, 402 third stage, and 260 in the fourth stage.

Sulfanilamide has been used in the treatment of these cases, as described by my[4] preliminary report. Three different methods of treatment were given: first, small doses (a maximum of 20 grains per day) orally in 25 cases; second, the small doses combined with the use of a saturated solution (0.8 per cent) dropped into the eyes in 905 cases and only local application in 385 cases. Seventy-one cases were given no treatment. Each patient was given a bulletin containing information about the spread of trachoma, the care of the eyes, and the necessity of discontinuing the tablets if any reaction occurred. They were also instructed to use the drops 8 to 10 times daily. In the 835 cases that have been rechecked, 105 noticed some reaction, such as nausea, rash, dizziness, or drowsiness. Fifty-three of these continued the tablets, but 52 stopped taking them.

The results in the first and second stages were the most satisfactory. Activity was arrested in 85 per cent of the first and 82 per cent of the second stage within 4 to 8 weeks (*Table 2*). In the third stage only 32 per cent were arrested, but an additional 62 per cent were greatly improved: 91 per cent of the fourth-stage cases placed on treatment were improved. Only 3.8 per cent of all stages reported no improvement. There were six cases which reported an acute flare-up of the infection while under treatment or after treatment had been discontinued. Julianelle[5] has reported many recurrences within 18 months after the arrest of the infection by oral sulfanilamide. An attempt is being made to check all of our cases at three- or four-month intervals for a period of two years. None of

STATISTICS OF CLINICS

Stages	Total No.	Rechecked Three Times	Rechecked Twice	Rechecked Once	Not Necessary	Not Possible	Not Contacted
1	381	82	66	96		80	57
2	343	54	46	81		84	78
3	402	78	91	104		74	55
4	260	32	34	69	123		2
Total	1386	246	237	350	123	238	192

Table 1

RESULTS OF TREATMENT

Stages	Activity Arrested		Improved		Unimproved		Total
	Number	Per Cent	Number	Per Cent	Number	Per Cent	
1	206	85	36	14.2	2	0.8	244
2	149	82	29	16.4	3	1.6	181
3	89	32	169	62	15	6	273
4			125	91	12	9	137
Total	444	63*	359	43	32	3.8	835

*Since no activity is present in the fourth stage, percentages are figured from total of first, second and third stages.

Table 2

the twenty-five cases on oral medication alone was arrested; however, over half of these cases were arrested when placed on the combined therapy. The results with local application alone and the combined therapy were the same in the first and fourth stages. The second and third stages responded better to combined therapy. There were 350 cases in need of operations of various kinds before a cure could be expected. It is hoped that the next State Legislature will make some provision for this very necessary part of any trachoma program.

The cost of this program might be of interest. During the time the work was under the control of the Welfare Department, 5,052 persons were examined at an average cost of 75 cents per examination. Out of this number 891 cases were placed under treatment, thus making an average cost of $4.37 for each patient treated.

The cost since the State Board of Health assumed control can be best judged by the month of September. This was the first month that the full-time clinician and nurse were in the field. The yearly budget, as set up at present, is $16,100. This gives a monthly budget of $1,342. During September, 1,699 persons were examined and 279 cases treated. This would indicate a cost of 79 cents per examination, or $4.80 per case of trachoma. This does not show the real value of a trachoma program in dollars and cents. Even if we forget the economic value of a seeing person, and the comfort and happiness brought to him and his family, we can still figure in cold cash the amount saved by the State. With 56 per cent of the third and fourth stages blind, we can expect that 405 of our first and second stage cases would be doomed to blindness if untreated. In these stages we have been able to arrest the disease in 83½ per cent of cases so that at least 338 persons have been saved from blindness. If we only presumed that 200 of these would receive blind assistance of $120.00 per year, for 20 years, we have saved the taxpayers $24,000 a year, or a total of $480,000 during the period.

SUMMARY

We have shown how Arkansas is administering the control of trachoma through ambulatory clinics. The cost of trachoma control in this manner is negligible compared to the economic saving.

The use of sulfanilamide orally and locally combined, or locally only, will arrest activity in 83.5 per cent of first and second-stage trachoma. Third- and fourth-stage trachoma may be greatly improved by the same treatment, and infectiousness can be eliminated.

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GUNSHOT WOUNDS OF THE PREGNANT UTERUS*

By T. C. Bost, M.D., F.A.C.S.
Charlotte, North Carolina

It seems strange that gunshot wounds of the pregnant uterus are so rare as to offer a kind of medical curiosity. Especially is this true when we consider the important place of firearms in the social affairs of the negro race and the great number of other gunshot wounds, intentional and accidental, among these people. Since during her period of possible childbearing the average negro women is pregnant about half the time, and since negroes live crowded together in small houses, and since there is hardly a gathering of them without a woman advanced in pregnancy's being present, it would be expected that the large target afforded would be oftener struck. And to this chance must be added that of accidental gunshot wounds and attempts at suicide in other races.

In a review of the literature I find mentioned eleven cases occurring previous to 1910, but since I can find no available information concerning the extent and outcome of these cases, I am unable to include these in my report. For the 30 years I have been able to find nine reported cases, not including the one I am here reporting, which brings the total to ten cases in 30 years. Four of these cases occurred in this country, the other six in a number of foreign countries. No mention is made of a case in the negro race.

Because of the small number of reported cases it is impossible to draw general conclusions. It would seem that each case must be decided on its own merits.

*Received for publication May 19, 1941.

These injuries are of great interest both to those doing obstetrics and those doing surgery. They differ from the usual gunshot wound in that two lives instead of one are at stake; and it is the general opinion that in any kind of disease or trauma and shock the patient's condition is made more hazardous by the state of pregnancy.

If a lesson may be drawn from the cases of which we have record, the reverse appears to be true, pregnancy apparently offering a kind of protective mechanism so that the mortality appears to be much less than the general mortality in gunshot wounds of the abdomen.

The indications for opening the abdomen would appear to be the same as though the pregnancy did not exist. The probability that other viscera may be injured is such that operation for exploration can scarcely be avoided, whatever may be the indication as to the effect on the womb and its fruit. By the same token the severity of the injury is determined chiefly by three factors: (1) the degree of visceral damage, (2) the amount of hemorrhage, and (3) the time elapsing from the time of injury to the completion of the surgical repair.

When the abdomen is explored and no serious injury to the viscera is found except to the uterus there is room, perhaps, for a difference of opinion as to subsequent treatment. The general opinion is that the uterus should be emptied if the uterine cavity has been penetrated. The first question, then, to decide is whether to empty the uterus by cesarean section, or to suture the wound or wounds in the uterus, close the abdominal wound and await the expulsion of the fetus. Fowler advised a cesarean section if the fetus is alive and so far along as to be likely to survive. He advises emptying the uterus from below unless there is already a large opening in the uterus through which it may be easily emptied and then repaired. The general opinion would seem to be that the uterus should be emptied by cesarean section at the time of exploration, at almost any stage of pregnancy; and most of the cases have been treated this way. In six of the ten reported cases, including my own, and these were in various different stages of pregnancy, cesarean section was done; in two cases hysterectomy was done "to arrest pelvic hemorrhage," one case was operated upon, the bullet was removed from the fundus of the uterus and the child delivered normally 17 days later; one case was not operated upon and labor came on and a living child was delivered normally three days later.

CASE REPORT

A housewife, aged 20, with two children, had never had serious illness and her health had been generally good. Menstruation was always normal. Her last period occurred in September, 1938. Since cessation of menstruation she had had much nausea and vomiting and slight vaginal bleeding, but had had no treatment for this. She did not want another child or to endure this pregnancy. She shot herself with a .22 rifle in the abdomen February 19, 1939, and was admitted to Mercy Hospital an hour later in mild shock complaining of pain in the epigastrium and right side, also of slight nausea. Her pulse was 100, temperature 98 degrees, and respirations were 24.

The patient was well developed and the pregnancy appeared to be of five months' duration. There was a bullet wound with powder burns just below the right costal margin and moderate rigidity and tenderness in the epigastrium and over the right side. There was no tenderness or rigidity of the left side, and no vaginal bleeding.

Immediate preparation was made for operation. While this was being done a flat x ray picture (*Fig. 1*) was made by Dr. Robert H. Lafferty, which he reported as follows: the bullet entered just below the right costal margin, course downward, is buried in the pubis. In the passage the distribution of lead fragments leads us to think that it touched the cranium of the fetus.

A catheterized specimen of urine was clear. White blood cells were 18,000; red cells 3,500,000; and hemoglobin 65 per cent.

At operation, ether was administered by the referring physician, Dr. Van Matthews. A midline incision to

Fig. 1
(1) Entrance of bullet. (2) Bullet lodged in pubes.

TEN CASES OF GUNSHOT WOUNDS OF THE PREGNANT UTERUS

Reported	Term of Pregnancy	Symptoms	Extent of Injury	Operation	Fetus Result	Mother Outcome
Fowler 1910 Brooklyn, N. Y.	8 mos.	Uterine contraction, very little shock, 3 hours after injury, no vaginal bleeding	Lover pressed revolver 32 against right side of abdomen. Bullet passed through uterus. No other visceral injury. Exit left side of abdomen	Cesarean section	Three fingers off right hand, recovered	Recovered
Fudge 1912 Elmire, N. Y.	8 to 9 mos.	Moderate amount of shock. Considerable hemorrhage from the wound. No vaginal bleeding	Attempted suicide rifle 32. Entrance right flank. Exit 1 inch left umbilicus. Uterus incised by bullet. Child expelled from uterus into abdomen.	Child removed from peritoneal cavity. Wound closed through which child was expelled	Through pelvis and spine, dead	Recovered
Tucker 1912 Shanghai China	9 mos.	12 hours after injury. Patient in marked shock and weak from great loss of blood	Shot by robber. Bullet entrance 3 inches above and to left of umbilicus. No exit. Entered at fundus of uterus. No other visceral injury.	Cesarean section. (No mention of transfusion)	Bullet lodged in back, recovered	Had sudden collapse on third day, thought to be hemorrhage, died
Belcher 1917 Northern France	? Far advanced	Three days after injury comfortable until labor came on	Rovolver bullet. Entrance in perineum near anus; ranged upward and lodged in uterus.	No operation. Labor came on and delivered three days later	Bullet in head, born dead	Recovered
Saint Goehlinger and Poier 1920 Paris, France	6 mos.	Very little shock. Moderate tenderness and rigidity. Vaginal bleeding	Shell gutter wound anterior abdominal wall and gutter wound of uterus. No other visceral injury	Cesarean section. Repair of uterus and anterior abdominal wall	Back injury, lived 15 hours	Recovered
Stiglbauer 1924 Vienna Germany	?	Six hours after injury, acute abdominal symptoms	Browning pistol 7.65 mm. Entrance left gluteal region and lodged in uterus. Three perforations small intestine, sigmoid. Pelvic hemorrhage	Suturing perforations and hysterectomy to control pelvic hemorrhage	?	Recovered
Placintiamu and Turcanu 1928 Bucherest Rumania	Last month	Uterine cramps; no symptoms of peritoneal reaction at first, 4th day pain in left side	Bullet wound entrance to right of umbilicus and lodged in fundus of uterus. No other visceral injury	Bullet removed from fundus uterus; child delivered normally 17 days later	Injury to left leg, recovered	Recovered
Motta Maia and Veanna 1929 Brazil	?	Moderate shock with symptoms of peritonitis 11 hours after injury	Bullet wound entrance mid epigastrium through fundus of uterus, two perforations of lower ileum	Cesarean section, perforations closed	Injury to child's arm, recovered	Recovered
Belknap 1939 Damariscotta Maine	6 mos.	After several hours mild symptoms of shock	Attempted suicide rifle 22. Entrance to left of umbilicus, through uterus and lodged in pelvis, pelvic hemorrhage	Hysterectomy to control pelvic hemorrhage	Wound of chest, dead	Recovered
Bost 1940 Charlotte N. C.	5 mos.	Pain in epigastrium. Moderate tenderness and rigidity, moderate shock; leukocytes 18,000; urine clear	Attempted suicide rifle 22. Entrance right costal margin, ranging downward and lodged in pubes. Two holes in ileum. Passed through uterus and two holes in bladder	Cesarean section, closing holes in ileum and bladder, indwelling catheter	No injury, lived 1 hour, premature	Recovered

Table 1

the right and below the umbilicus was made, since it was apparent that the injury was in the lower abdomen, although the entrance wound was rather high. There were several ounces of blood in the abdominal cavity. Two holes were found in the uterus, one in the fundus and the other in the lower segment anteriorly. A cesarean section was done by incising the area between the two uterine wounds, which were about five inches apart. Removing the few blood clots and some free blood, the fetus and membranes were found to be intact. The uterus was emptied and sutured. Search disclosed two perforations in the lower ileum two feet from the ileocecal valve, which were closed with 00 chromic catgut; and two in the dome of the bladder, which were closed and a drain put in this area. A self-retaining catheter was put in the bladder. Recovery was uneventful and the patient was dismissed from the hospital on the fourteenth day. The fetus was not injured by the bullet, but it lived only about one hour, being about five months premature.

This patient again became pregnant and an elective cesarean section and sterilization were done by Dr. Van Matthews May 14, 1940, thirteen months after the injury. Both mother and child made a good recovery and are now in good health.

SUMMARY

Of the prospective mothers whose cases are reported (*Table 1*) three, including my own, attempted suicide with rifles. They all recovered. One woman was shot by her lover; two were victims of war wounds, one from a shell fragment and the other from a pistol ball; one was shot by a burglar. In the other four cases the manner of injury was not stated.

In seven cases there was no visceral injury other than that to the uterus. In these cases perhaps the force of the missiles was so used up in the pregnant uterus as to prevent disastrous results to other viscera. Also in these seven cases the enlarged uterus apparently offered further protection in filling the lower abdomen and forcing the intestines out of range of the missile. In three cases there were intestinal perforations; in my own case, bladder perforations in addition. This was the only bladder injury in the series.

Nine mothers recovered and one died, a mortality of 10 per cent. Although this series of cases is small, yet it tends to show a remarkable contrast to the general mortality of gunshot wounds of the abdomen, which is 30 to 70 per cent or even higher.

Of the six viable children four recovered and two died, a mortality of 33 per cent.

The case here reported, together with the other reported cases, would tend to show that pregnant women can tolerate violent trauma, that the pregnant uterus itself is very resistant to both trauma and infection, as no mention was made of puerperal sepsis in any of these cases.

This case also illustrates the importance of a flat x-ray picture when there is no exit wound to determine the course of the bullet and the possible injury, so that the proper incision can be predetermined. Also, that a negative urinalysis report does not rule out bladder injury.

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CAN THE SITE AND DEGREE OF INJURY AND THE PROGNOSIS IN HEAD TRAUMA BE ACCURATELY DETERMINED BY SPINAL FLUID ERYTHROCYTE COUNTS?*

AN EXPERIMENTAL STUDY

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INTRODUCTION

There are still many unsolved problems concerning the mechanism and nature of the pathologic processes that develop following head trauma.

Two years ago, experiments which were conducted on dogs with reference to the disappearance of erythrocytes from the cerebrospinal fluid after their introduction into the cisterna magna, convinced us that, following the first forty-eight hours after the introduction of a measured

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amount of blood into the dog's cisterna magna, only 4.5 per cent or less of the red blood cells introduced could be recovered by complete subarachnoid drainage (by means of lumbar laminectomy). After the first forty-eight hours, nearly all the red blood cells had disappeared from the spinal fluid spontaneously by hemolysis or otherwise. These original experiments[1] substantiated a clinical impression of ours, namely: that so far as removal of erythrocytes is concerned, lumbar puncture is of slight value and should not be done for the sole purpose of eliminating them from the cerebrospinal fluid.

Our attention was next directed to the experimental problem: can the site and degree of injury and the prognosis in head trauma be accurately determined by spinal fluid erythrocyte counts? The present report has to do with the results and interpretation of experiments directed toward the solution of this question.

It has long been customary, in cases of head injury, to utilize the erythrocyte count of the spinal fluid as one of the guides for prognosis and for estimating the site and degree of the trauma. Our clinical observations of certain post-traumatic brains, seen at the autopsy table, together with the antemortem clinical and laboratory findings in such patients have, in many instances, not been in accord with these opinions, as a lack of correlation is occasionally demonstrated between antemortem cerebrospinal findings and the actual injury as seen at autopsy.

Many clinicians believe that a very bloody spinal fluid after head injury is probably indicative of basilar bleeding (similar to that which occurs following a ruptured basilar aneurysm in spontaneous subarachnoid hemorrhage) or intraventricular hemorrhage, with a relatively poor prognosis. Some observers record the erythrocyte counts of the spinal fluid in all cases of intracranial trauma and believe that the higher counts are of definitely unfavorable prognostic significance; this has been stated by Coleman,[2] Connors and Wright,[3] and by Munro,[4] and is undoubtedly true in certain cases. Munro and Maltby[5] have quite recently (1941) emphasized that the danger of lumbar puncture in head injuries is minimal and that the information obtained is not only essential but of a nature that far outweighs any possible risk. They definitely implied, in commenting on cases of extradural hemorrhage that showed bloody cerebrospinal fluid on lumbar puncture, that the more red blood cells present in the cerebrospinal fluid, the greater the brain damage; that is, that a direct correlation necessarily exists. This diagnostic employment of the number of erythrocytes in the cerebrospinal fluid is further demonstrated in Leary's[6] statement in describing a case of extradural hemorrhage: that the finding of a grossly clear lumbar puncture fluid should have suggested that there was no damage to the brain or arachnoid. That such reasoning is not necessarily correct is shown by the experiments to be described and by two of our clinical cases to be cited in this report. All clinicians are familiar with patients with very great numbers of red blood cells in the spinal fluid following head trauma who made complete asymptomatic recoveries. On the other hand, a patient in our hospital who had had a bursting fracture of the skull due to a fall from a height showed, on postmortem examination, extensive brain laceration with intracerebral and subarachnoid hemorrhage, death having occurred about fourteen hours after injury. However, the cerebrospinal fluid from the lumbar sac, studied several hours before death, showed no erythrocytes. We have also recently treated a patient who had a massive right (unilateral) intraventricular hemorrhage incident to a minor head injury. There were only a few red blood cells per cubic millimeter in the spinal fluid (which was grossly quite clear) and in the left lateral ventricular fluid; however, the right lateral ventricular fluid contained a large liquid clot which was evacuated with eventual recovery of the patient. Coagulation of the hemorrhage must have occurred spontaneously at the right foramen of Monro; a ventriculogram showed air in the left lateral and third ventricles only, both of which were displaced to the left side. These two clinical cases alone clearly demonstrate that severe contusion and laceration of the brain with resulting extensive subarachnoid bleeding or massive intraventricular hemorrhage are not necessarily accompanied by a grossly bloody spinal fluid.

EXPERIMENTAL METHOD

Twelve dogs were used in the experiments. Each one was anesthetized with sodium iso-amyl ethyl barbiturate administered intraperitoneally (50 mg. kilo of body weight).

After anesthetization, a button of bone was removed with a hand trephine over a cerebral hemisphere without injury to the dura. A cisternal puncture was then performed with a No. 20 gauge needle and the cisternal fluid was immediately examined to be certain that there were no red blood cells present from needle trauma. A sharpened bent wire was introduced through the dura into the cerebral cortex (to

the same depth in each dog) and rotated three times through 360°. The same wire was used in all the experiments so that the lacerations produced were as nearly uniform as possible. There was no appreciable loss of cerebrospinal fluid during the introduction and rotation of the wire. Immediately on withdrawal of the wire, the button of bone was accurately replaced and sealed with bone wax around its periphery. Samples of cisternal fluid (1 c. c.) were removed at twenty-minute intervals thereafter through the previously placed indwelling cisternal needle (the lumen of the needle being tightly corked after the removal of each specimen of fluid) in most instances up to four hours and twenty minutes after induction of the trauma, following which the animal was sacrificed by a formalin injection into the heart. Each brain with its intact membranes was immediately removed and placed in 10 per cent formalin for further study. The individual cisternal fluid samples were examined as to their erythrocyte content and these findings were later correlated with the site and degree of trauma as determined when each individual formalinized brain was examined on its intact superior and inferior surfaces and after transverse sectioning of the brain at intervals of about one centimeter.

RESULTS

The results demonstrate that the site and degree of an intracranial hemorrhage cannot be estimated accurately or even approximately by the absolute number of red blood cells in the spinal fluid at any time, nor can the prognosis be determined in any individual case by the number of erythrocytes in the cerebrospinal fluid if such a prognosis is based on the probable location and degree of the bleeding for, in many instances, there is no correlation demonstrable.

The total number of red blood cells in the cisternal fluid, as expected, varied greatly in the different experiments (although the type and degree of induced trauma was the same) due to the wide difference in vascular architecture of the individual brains.

After careful scrutiny of the twelve brains on their intact superior and inferior surfaces, and on sectioning, the specimens were arranged, for purposes of easy demonstration and comparison, into four groups. *Fig. 1* illustrates a single brain (Dog A), shown by itself, as it represents the greatest number of cisternal erythrocytes that were recovered from any dog in the entire group (*Chart 1*). It is noteworthy that in this brain there was no intraventricular bleeding (the arrow points to the ventricular system) or basilar hemorrhage, an intracerebral extraventricular clot and subarachnoid bleeding over the left cerebral convexity constituting the only significant hemorrhage.

Fig. 2, the second group, illustrates four dogs' brains, constituting the animals in which the major if not the entire amount of bleeding occurred into the ventricular system, either bilaterally (Dogs B and C) or in one lateral ventricle only (Dogs D and E). By referring to *Chart 1*, in which the number of cisternal erythrocytes is plotted against time intervals of twenty minutes, it is seen that there is a wide difference in the number of red blood cells from each individual dog, although each one had gross bleeding in one or both lateral ventricles. Dog B, with a massive bilateral intraventricular hemorrhage, had the second highest number of red blood cells in the cisternal fluid of all the dogs studied, but definitely less than did Dog A (*Fig. 1*) with no intraventricular or basilar hemorrhage at all. Furthermore, although Dog B (*Fig. 2*) was the only animal to die before the conclusion of the experiment, it did not succumb (*Chart 1*) until the red blood cell count was definitely receding from its peak which occurred one hour and twenty minutes after the induction of the trauma. Dog C (*Fig. 2*), also with a massive bilateral intraventricular hemorrhage, and with little or no subarachnoid bleeding, had one of the highest red blood cell counts of any of the dogs (*Chart 1*), but here again, definitely less than did Dog A with no intraventricular or basilar hemorrhage.

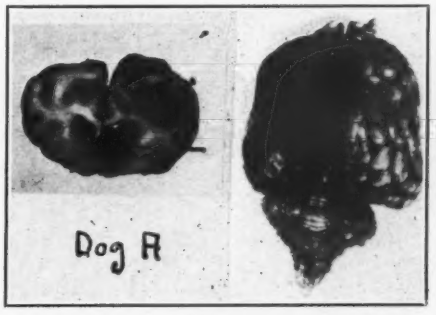

Fig. 1

Dog A, shown individually as it represents the highest number of red blood cells in the cisternal fluid found in any of the twelve dogs studied (see Chart 1). Notice the absence of gross bleeding into the ventricles or basilar cisternae. The arrow in the left hand figure points to the choroid plexus in the left lateral ventricle.

N. B.: The cross section of the brain in this specimen, as well as in the other eleven specimens (Figs. 2, 3 and 4), is the one showing the maximum amount of hemorrhage in each individual brain.

It is to be emphasized that Dogs D and E (*Fig. 2*) with definite unilateral ventricular hemorrhages had surprisingly low erythrocyte counts in the cisternal fluid (*Chart 1*), thus resembling, although not to so complete a degree, the second clinical case previously cited in the introduction of this paper.

Fig. 3, also a single brain (Dog F), is illustrated separately, as it constitutes a good example of massive hemorrhage into the basilar cisternae without other appreciable bleeding except slight intracerebral extraventricular hemorrhage at the site of the induced trauma. Reference to *Chart 1* clearly shows that this massive basilar hemorrhage, presumably in full and direct communication with the spinal subarachnoid space, was accompanied by surprisingly few red blood cells in the cisternal fluid at any time.

Fig. 4 illustrates the miscellaneous remaining group of six brains (Dogs G, H, I, J, K, L) in which the hemorrhages occurred, for the most part, intracerebrally (extraventricularly) or over the superior cerebral convexities. In all but one, these hemorrhages were associated with low red blood cell counts in the cisterna magna. However, Dog K, with very little bleeding, as shown in the three views of the brain (*Fig. 4*), was exceeded only by Dogs A, B and C (with massive bilateral intraventricular or subarachnoid hemorrhage) in the number of red blood cells in the cisternal fluid (*Chart 1*). So far as could be determined, and this applies to all the dogs, none of the cisternal bleeding could be attributed to the indwelling cisternal needle itself; the bases of the brains and the cisternal regions were carefully inspected at postmortem examination to eliminate this possibility.

A totally unexpected but intriguing finding (with one notable exception) was the fact that in almost all the dogs (83.3 per cent) the height of the bleeding into the cisterna magna, regard-

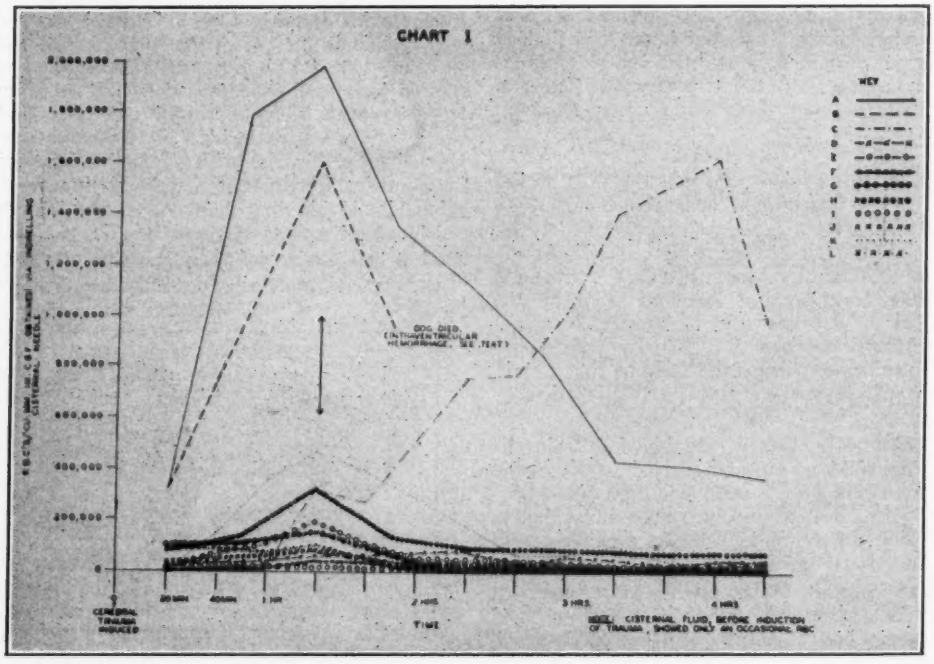

Chart 1

Composite curve of the twelve animal experiments. The number of red blood cells per cu. mm. appearing in the cisternal fluid of each dog is shown at twenty-minute intervals after induction of the trauma. Dog A (Fig. I) with no intraventricular or basilar hemorrhage had the highest number of red blood cells in the cisternal fluid. Dogs D and E (Fig. II) with massive unilateral ventricular hemorrhage, had relatively few red blood cells in the cisternal fluid. Dog F (Fig. III) with a massive hemorrhage in the basilar cisternae, had very few red blood cells in the cisternal fluid. Dog K (Fig. IV) with very little bleeding anywhere as shown in the postmortem specimen, had the fourth highest red blood cell count in the cisternal fluid of the twelve animals studied. Curiously enough, the height of the bleeding in practically all the dogs, regardless of the site or degree of the trauma, occurred one hour and twenty minutes after the injury was induced.

less of whether the number of red blood cells in the cisternal fluid was slight or great and irrespective of the intracranial location and degree of the hemorrhage, occurred one hour and twenty minutes after the trauma was induced (*Chart 1*).

DISCUSSION

The clinical cases already cited and the experimental data herein described lead to the conclusion that the absolute number of red blood cells in the spinal fluid after head injury is not invariably an accurate criterion as to the site or degree of intracranial bleeding nor is it therefore a reliable guide to prognosis in any individual case.

The greatest number of red blood cells in the cisternal fluid of all twelve of the experimental brains examined after an induced trauma was found in a dog (A, *Fig. 1*) with subarachnoid bleeding over the cerebral convexities and intracerebral (but extraventricular) hemorrhage and with little or no basilar bleeding. Therefore, the prognosis should be relatively good, even though the cisternal fluid of this dog contained more red blood cells than any of the others. A reasonable explanation for the finding of relatively few red blood cells in the cisternal fluid following massive intraventricular or basilar subarachnoid bleeding in certain cases is probably that of Bagley,[7] who showed that agglutination and coagulation of erythrocytes in the subarachnoid spaces occur rapidly in intracranial bleeding, with a resultant block so far as obtaining red blood cells below the site of the hemorrhage is concerned. Coagulation at the foramina of Monro, in ventricular hemorrhage, accounts for other low red blood cell counts in the spinal fluid. Spontaneous hemolysis also undoubtedly explains the elimination of a considerable number of the erythrocytes, even after

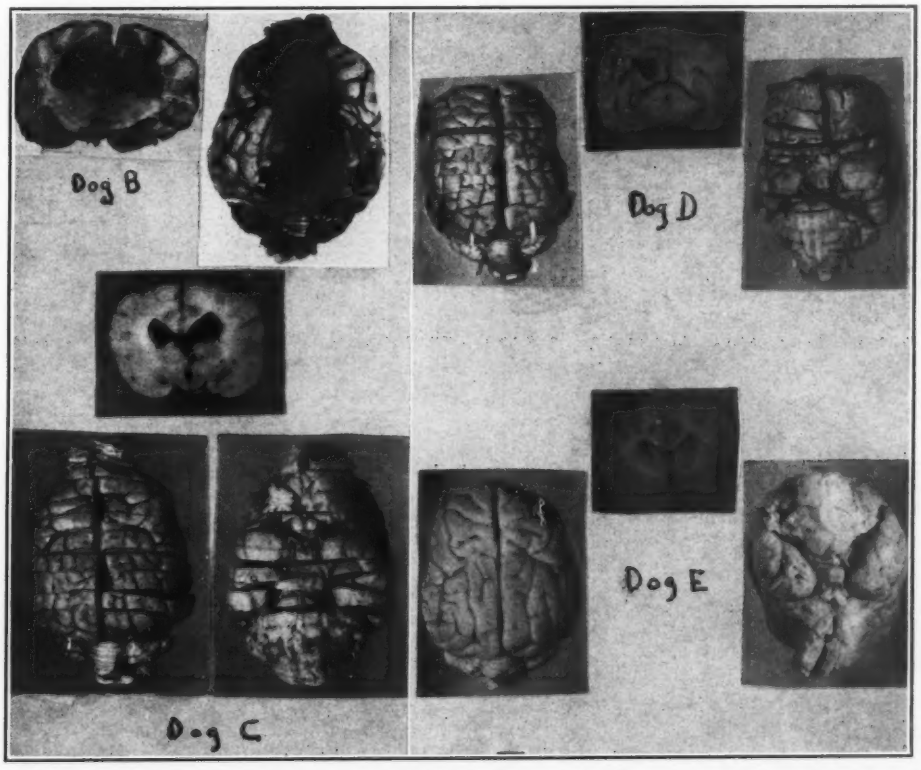

Fig. 2

Dogs B, C, D and E (predominantly ventricular hemorrhages). Specimens D and E, each containing a massive unilateral ventricular hemorrhage, showed surprisingly few red blood cells in the cisternal fluid (Chart 1); spontaneous coagulation at the foramen of Monro or in the third ventricle must have occurred.

so short a time interval as several hours after the trauma is induced; such hemolysis may occur more rapidly in some individuals than in others.

In considering prognosis, it is possible that fatal outcomes in cases of massive subarachnoid hemorrhage alone may be due to greatly increased intracranial pressure (rather than to the amount of blood *per se*) incident to the outpouring of blood into the subarachnoid space, as shown by Parker and Lehman.[8] They found (1936), in animal experiments, that there is a direct correlation between the cerebrospinal fluid pressure and the amount of bleeding into the subarachnoid space, whereas comparison between the cerebrospinal fluid pressure and the amount of intracerebral bleeding shows no such correlation.

A red blood cell count taken in the case of Dog B just before death might have led one to give a more favorable prognosis than would have been justified, if one assumes, as is so often done clinically, that the higher the red blood cell count the graver the prognosis at any particular time; in this instance the number of red blood cells was diminishing sharply up to the point at which the dog died (*Chart 1*).

It should be emphasized that these experiments are not directly applicable to a study of prognosis, as the animals were sacrificed at the end of a four-hour and twenty minute period (at which time bleeding was at a minimum or definitely subsiding and the dogs were in good condition with the exception of Dog B, previously cited) and hence one does not know how long each individual dog might have survived. However, the experiments do tend to disprove the clinical tendency of suggesting a grave prognosis if there are large numbers of red blood cells in the spinal fluid. A comparison of *Chart 1* with the illustrations of the individual brains makes it clear that some of the severe brain injuries are accompanied by considerably less numbers of red blood cells in the cisternal fluid than are others (such as Dog K) whose lesions, pathologically, appear rather insignificant.

Levinson[9] stated that clear cerebrospinal fluid does not necessarily exclude intracranial hemorrhage, especially into the ventricles, and Strauss, Globus and Ginsburg[10] observed that occasionally the spinal fluid in cases of spontaneous subarachnoid hemorrhage, as proven by postmortem examination, even in certain patients with an abrupt and explosive onset, is only xanthochromic or may be clear. The importance of such statements as these should be more widely appreciated.

On the other hand, in one dog (B, *Fig. 2*) with a massive bilateral intraventricular hemorrhage, very large numbers of red blood cells were found in the cisternal fluid, although not to the

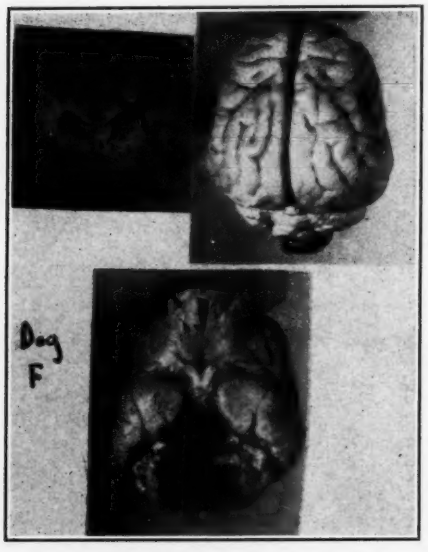

Fig. 3

Dog F, shown separately as it is the best example of a massive hemorrhage into the basilar cisternae. Although a large number of red blood cells would be expected in the fluid of the cisterna magna of this animal, an inspection of Chart 1 will demonstrate that, actually, very few erythrocytes were recovered at any time after the induction of the trauma until the conclusion of the experiment; in fact, next to the lowest number of any of the twelve dogs utilized for this study.

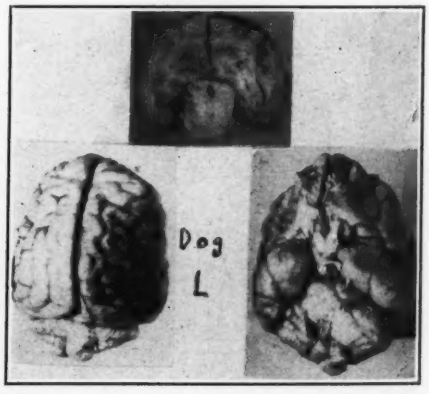

Fig. 4 (3)

See Fig. 4(2), next page, for legend.

degree of the first dog (A, *Fig. 1*) cited, that had superior cortical subarachnoid and extraventricular (intracerebral) bleeding only. Both of these dogs, however, had over a million and a half red blood cells per cubic millimeter in the cisternal fluid at the height of bleeding and, in them, no block or coagulation of erythrocytes, to any appreciable degree, apparently occurred. It is therefore impossible to state that a high red blood cell count in the spinal fluid after head injury probably indicates an intraventricular or basilar hemorrhage (with a relatively poor prognosis) rather than bleeding over the cerebral convexities or intracerebrally (extraventricular) only.

Dog C (*Fig. 2*) approaches, perhaps, the clinical problem of post-traumatic intracranial hemorrhage most closely. In this brain (*Chart 1*) there is seen, apparently, a secondary hemorrhage reaching its height four hours after the induction of the trauma. However, even in this brain with a massive bilateral intraventricular

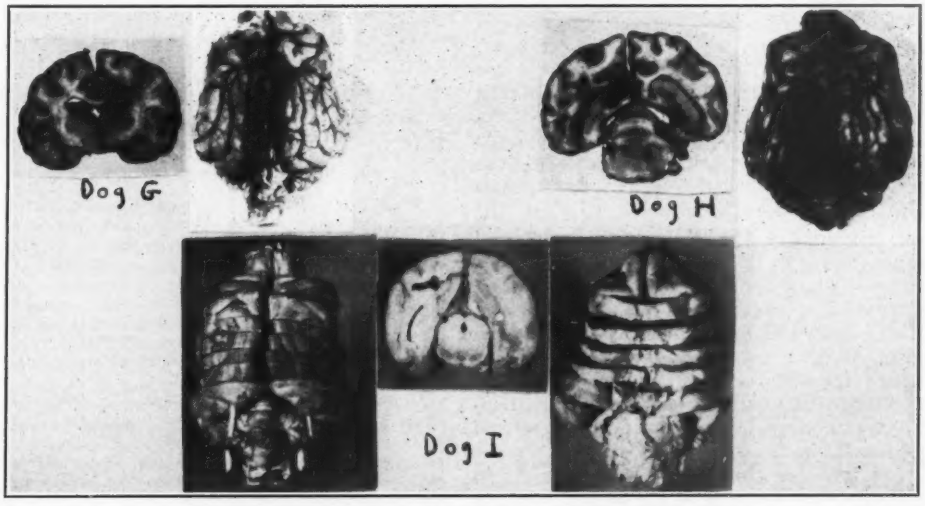

Fig. 4 (1)

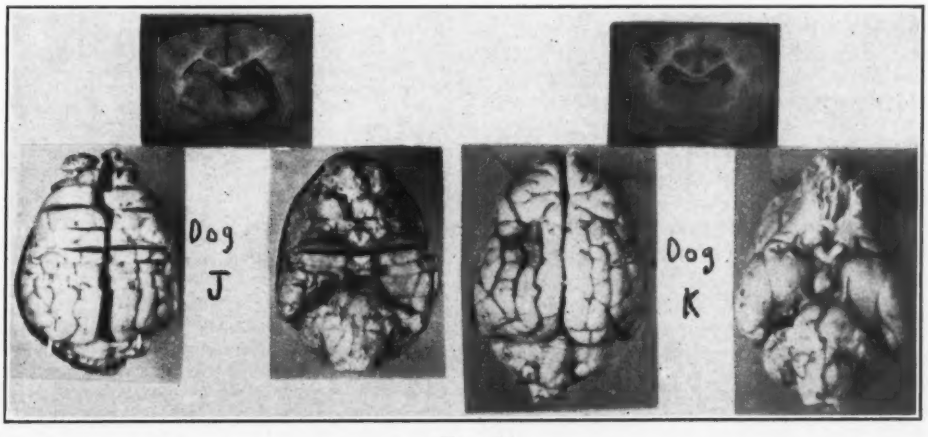

Fig. 4 (2)

Dogs G, H, I, J, K and L. A miscellaneous group of brains, most of which show relatively slight evidence of hemorrhage compared to the brains shown in Figs. 1, 2 and 3. However, Dog K, with relatively little bleeding in any location, had the fourth highest red blood cell count in the cisternal fluid of the twelve animals studied (Chart 1).

hemorrhage (*Fig. 2*), there were considerably fewer red blood cells at any time in the cisternal fluid than in Dog A (*Fig. 1*) with no intraventricular or basilar bleeding, demonstrating again the futility of trying to predict, solely from the number of red blood cells in the spinal fluid, how severe or in what location the bleeding has occurred.

It is not necessary to emphasize that one must be cautious about drawing conclusions from such a small number of experiments, but the results certainly appear to support the opinions just stated.

CONCLUSIONS

(1) The absolute number of red blood cells in the spinal fluid after head trauma is not necessarily an accurate guide to (a) the site of the intracranial hemorrhage, (b) the degree of the intracranial bleeding and therefore (c) prognosis, in any individual case.

(2) Experimentally, the greatest number of red blood cells in the spinal fluid, after the induction of a uniform brain injury in twelve dogs, was found in a brain free from intraventricular or basilar hemorrhage. Bleeding chiefly into the basilar cisternae may be accompanied by surprisingly few red blood cells in the spinal fluid. Unilateral ventricular hemorrhages, in particular, may produce relatively small numbers of erythrocytes in the spinal fluid, presumably due to coagulation at the foramen of Monro, or in the third ventricle. One of the largest numbers of red blood cells in the spinal fluid after experimental brain injury was found associated with a brain that showed, on postmortem examination, little or no evidence of subarachnoid, intracerebral, intraventricular or basilar hemorrhage.

(3) An unexpected but interesting finding in these experiments was that in the great majority of cases (ten out of twelve, 83.3 per cent) the maximum bleeding into the cerebrospinal fluid occurred one hour and twenty minutes after induction of a standard trauma of the dog's brain, regardless of the degree or location of the intracranial bleeding. This arouses an interesting speculation as to the optimum time for treatment of certain cases of post-traumatic intracranial hemorrhage.*

Note.—*This investigation was suggested by Dr. C. C. Coleman. The writer is also grateful to Dr. E. P. Lehman for valuable criticism and advice during the progress of the experiments, and to Dr. William Bray, Director of the Clinical Laboratories at the University of Virginia, who kindly provided all necessary facilities for the erythrocyte calculations of the cisternal fluids carried out by the writer.

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DISCUSSION (*Abstract*)

Dr. R. Glen Spurling, Louisville, Ky.—Dr. Meredith's experimental data support completely my notion about the value of the erythrocyte count in the spinal fluid as an indication of the severity of the cerebral damage.

I suppose there are those holding a contrary view who would find fault with his experiments, and I think these experiments may be justly criticized in that his experimental lesions are all intracerebral and, therefore, do not reproduce the same set of conditions which prevail with generalized cerebral trauma.

In my experience, the effectiveness of withdrawal of blood by spinal puncture in the treatment of patients with irritative meningeal symptoms following head trauma is quite different from that of Dr. Meredith. Many patients, anywhere from the third to the seventh day following trauma, develop severe irritative meningeal symptoms from blood. Repeated lumbar punctures during this period produce dramatic improvement of symptoms and I am sure hasten convalescence. Whether this amelioration of symptoms is due to withdrawal of red cells from the spinal fluid or whether other factors must be considered, such as the change of pressure, is something which has not been clearly proven. Certainly, from a clinical point of view the drainage of bloody fluid in the treatment of irritative meningitis from blood is a very useful procedure.

Dr. Meredith has made a valuable contribution to the subject of head trauma.

Dr. Franklin Jelsma, Louisville, Ky.—Dr. Meredith has given us the results of some experimental data based upon the problem, can the site and degree of injury and the prognosis in head trauma be accurately determined by spinal fluid erythrocyte counts?

He has decided the question in the negative, and clinically I think we can all agree with him that this cannot be done. Yet he has given us very definite and valuable material from which to draw conclusions.

In regard to the site of the injury, I would take it to mean whether the injury is cortical or intraventricular. Is the bleeding occurring from the cortex or from the ventricle? Certainly he has shown by his experiments that blood coming from either the cortex or the ventricle

can block off and not occur in the lumbar spinal fluid in some cases, while in others it will.

With certain clinical signs and without any spinal fluid blood at all, an extradural hemorrhage can be determined. It would be a very valuable asset to know whether there were any red cells in the spinal fluid in either an extradural hemorrhage or in a subdural hemorrhage.

The site of injury can probably be better determined by focal cerebral signs such as paralysis, aphasia, and so forth, all of a very definite clinical nature.

In regard to the degree of injury and prognosis, it is very difficult to determine either of these by the red cell count. In some of the most severe injuries, as Dr. Meredith has shown, there occurs very little bleeding, whereas in some of the less severely injured individuals more bleeding occurs, either from the ventricle or from the cortex.

There are other factors such as edema, venous congestion, subcortical hemorrhage and actual destruction of brain tissue, which are very important in determining the prognosis and the degree of injury, and these, I think, will have to be determined by clinical factors.

Dr. Meredith (closing).—The fact that some of the more severe intracranial hemorrhages do not show much blood in the spinal fluid when lumbar puncture is performed is certainly true. On the other hand, a patient with a very bloody spinal fluid may be profoundly unconscious, and obviously has a poor prognosis. Again, the patient who has a grossly clear spinal fluid, and yet clinically has a severe injury, has a poor prognosis, as emphasized by Dr. Jelsma.

One should be guided a great deal by the focal neurologic signs in determining the necessity for operation. For instance, the advent of localizing signs in the pupils and the extremities (unilateral weakness or convulsions and reflex inequalities), together with an increase in the degree of stupor, and general signs of increased intracranial pressure, chiefly slowing of the pulse and respiration, demand operative intervention, with the probability that one will find a subdural or an extradural clot.

Dr. Spurling referred to clinical cases as contrasted to the experimental type. We have found, in the last few years, in analyses, on three or four occasions of several hundred patients with head injuries of all types that were admitted consecutively to the hospital (without selection) that is, with concussions and all types of the more severe injuries, that in only about 15 per cent of each one of these separate groups that were analyzed did we resort to lumbar puncture.

We reserve lumbar puncture, as a therapeutic agent in the treatment of patients with head injury, for the occasional patient who does not do well clinically, who has severe headache, whose sensorium is clouded, and who does not have the classic subdural or extradural clot syndrome or who has had the possibility of such clots eliminated by previous operation. One will find that such a patient undoubtedly is improved and his sensorium cleared following lumbar puncture and the removal of 10-15 c. c. of spinal fluid.

The lumbar puncture that one does clinically is not performed altogether for the removal of erythrocytes, because we believe that (from our previous experiments cited at the beginning of this paper) after the first day or two practically all of the red blood cells are spontaneously hemolyzed or otherwise eliminated provided the hemorrhage has been instantaneous and not continuous. The experimental animal is not entirely analogous to clinical cases (as is often true) because in patients with brain injury there may be continuous intracranial bleeding over a period of days and not from one or two loci only as in the experimental animal, but, perhaps, from large areas in the brain.

Furthermore, concerning the utilization of lumbar puncture for the relief of the patient's symptoms, all of us, I am sure, have seen individuals who had a considerable amount of blood in the spinal fluid a day or two after injury; then, a week or more afterward, perhaps, another lumbar puncture was done and the blood had disappeared (at least grossly) from the spinal fluid, but the spinal fluid pressure may have been a great deal higher than at the time of the early lumbar puncture when there was gross blood in the fluid.

The probable explanation for this is that hemoglobin, as it disintegrates, produces a much higher osmotic tension in the spinal fluid, and therefore much more fluid is soon found in the subarachnoid space: one then observes a considerably higher spinal fluid and intracranial pressure temporarily. Certain patients may be greatly relieved of headache and other symptoms at such periods in their convalescence by the reduction of the increased intracranial pressure *per se*, through the withdrawal of fluid by lumbar puncture rather than by the removal of the red blood cells themselves.

ANESTHESIA FOR INTRACRANIAL SURGERY*

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One or more of the serious difficulties which beset anesthetists may arise during anesthesia for any type of case, but it has long been realized that in no group of cases do so many of these difficulties gather together as during operations upon the brain. In recognizing the potentialities for trouble during intracranial operations, Mennell[1] felt in 1930 that the use of a form of endotracheal anesthesia offered the best defense against it.

In developing our present routine for the handling of the anesthesia for intracranial surgery we have brought to our aid three prominent developments of fairly recent years, in addition to the fundamental use of ethylene or nitrous oxide gas as an inhalation agent.

Although endotracheal instrumentation and insufflation are not recent developments,[2] it was as late as 1909 that Elsberg[3] based his clinical application of the early insufflation of ether

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vapor for anesthesia on the animal experiments of Meltzer and Auer.[4] During the decade following the World War, a number of men experimented with this method of anesthesia. Notable in this group was Magill,[5] [6] whose large-bore soft-rubber endotracheal catheter was the simple basis for the American modifications of Waters,[2] Guedel, Tovell,[7] Hargrave[8] and others. Our modification of Magill's tube is described herein. The endotracheal method has become widely accepted as indicated by numerous reports such as that of the Section on Anesthesia for the Mayo Clinic,[9] [10] where over 10,000 cases have been anesthetized by this method in the last ten years. Occasional accidents[11] or complications[12] do not detract from the value of properly employed endotracheal technic.

The clinical introduction of carbon dioxide absorption by Waters[13] in 1924 gave us a means of closing the patient's respiratory system with a preservation of warmth and moisture and quieter breathing, all of which show their advantage most in long operations. This method has been applied to all types of cases and to all inhalation agents.

Since Dr. Kreiselman[14] gave his first dose of tribromethyl alcohol (avertin) in 1928 we have used this agent more than 16,000 times. At Emergency Hospital alone, between July 1, 1932, when I entered the hospital as an intern, and July 1, 1940, we used it 13,498 times. Our associates, Bruner and Jackson,[15] are reporting elsewhere this series of cases with a full discussion of our technic in handling tribromethyl alcohol (avertin) patients and the restrictions and limitations which we place upon the use of this drug. Because of this very extensive experience, no effort is made here to summarize the numerous papers in the literature, quite a few of which[16] [17] [18] [19] [20] point out the advantages of this drug in neurosurgery.

We have always supplemented tribromethyl alcohol (avertin) with ethylene except in the presence of a known explosion hazard such as during the use of x-ray apparatus, the electrosurgical unit or even the actual cautery, in which cases we have used nitrous oxide.

In using ethylene for fifteen years, we, like Woodbridge,[21] have found it to be the safest and least toxic of general anesthetic agents. We have felt that particularly in long cases it offered many advantages over ether and that these advantages were especially noticeable in brain operations where cerebral edema and increased intraventricular pressure are caused by prolonged ether administration.[22] When attended by no degree of anoxia, nitrous oxide administration has most of the advantages of ethylene.

The safety of procaine infiltration gives certain theoretical advantages to this form of anesthesia for brain surgery. However, the duration of these operations makes them into trying ordeals for conscious patients. Also the surgeon must worry constantly about the general condition of the patient, often to the detriment of his surgery. Therefore, we prefer general anesthesia for these cases where any comparable degree of safety can be expected.

With the background in general anesthesia outlined above, it was only natural that we should adopt our present routine for anesthesia in intracranial surgery. This routine has been used in part by many[8] [16] [17] [18] [19] [20] [23] [24] and a few[22] have followed it almost completely.

Since the conduction of anesthesia is even more important than the selection of method, a detailed discussion will be given of our routine for the past five years.

Any carefully designed technic must be scrupulously followed to be most effective. Meticulous attention to detail and unhurrying performance of every step in procedure are nowhere more rewarded than in anaesthesia for intracranial surgery.

The preoperative preparation of these patients is variable and quite individualized because of the great variation in their general condition. The anesthetist should learn as much as possible about each case preoperatively. Practically all of them receive a moderate sized dose of morphine sulfate one hour before operation. Tribromethyl alcohol (avertin) is given about thirty minutes before operation and this dosage is varied according to several important factors.

We have little patience with the statements which pervade almost the entire literature on tribromethyl alcohol (avertin) anesthesia, that a certain dosage such as 90 mg. per kilogram of body weight is used. In the administration of any depressant medication, whether it be barbiturate, narcotic, or tribromethyl alcohol (avertin), Guedel's concept[25] [26] of the parallelism between metabolism and reflex irritability is a most important one. When tribromethyl alcohol (avertin) is administered on this basis the largest dose per unit of weight will be three or even four times the smallest one. Our actual range of dosage producing sleep deep enough for a pharyngeal airway to be kept in place has been from 35 to 150 mg. per kilogram of body weight.

Although it is true that more than two-thirds of our cases have received from 80 to 95 mg. doses, it is most important that some of them approach the extremes of the total wide range.

To develop further the metabolism—nerve irritability parallelism, we constantly make use of these facts: normal metabolism increases rapidly from the low level at birth to its highest peak at six or seven years, following which there is a slightly prepubertal drop. A secondary, but slightly lower, peak occurs at the end of the major changes of puberty after which metabolic rate gradually declines to an old age level as low as that at birth. Although any individual will tend to fall on this curve according to his age, he may be thrown above or below it by one or more of the factors which at any given time will influence general metabolism. Endocrine status, such as hyperthyroidism, or general bodily strength and total size, may influence the relation to normal metabolism for relatively long periods of time. On the other hand, emotional disturbances, pain, and fever, though more temporary, can cause marked deviations from average. A given patient is placed on the curve in his age level and then his exact position estimated in terms of the foregoing six factors. With his curve position known, tribromethyl alcohol (avertin) or other depressant dosage can be computed rather accurately as vector quantities measured to a metabolic base line somewhat below the birth-senility level. In this manner, a child of six on the normal curve would receive 100 mg. of tribromethyl alcohol (avertin) per kilogram of body weight and probably go deeply enough to sleep to tolerate a carefully inserted pharyngeal airway. However, if this child's curve position were 15 to 20 per cent above normal, he would require 115 mg. per kilogram of body weight, while if debility or narcotic pre-medication had placed him 15 per cent below normal, only 85 mg. per kilogram of body weight would be sufficient. A 50-year-old patient on the normal curve would receive 70 mg. per kilogram dosage.

After a dose of tribromethyl alcohol (avertin) selected on this scale, the patient is usually deeply enough asleep to tolerate a pharyngeal airway in 15 minutes. If not asleep, ethylene-oxygen is given for a few minutes before 10 per cent cocaine is applied directly to the larynx, cords, and proximal trachea by a cotton swab through a Jackson type laryngoscope. This usually causes moderate coughing which is quickly stopped by additional ethylene-oxygen through a face mask. The laryngoscope is used for this step because it allows cocaine to be applied accurately to the areas selected, requires the smallest amount of any method, and because it aids selection of the best tube for the intubation. Two or three minutes of ethylene anesthesia after cocainization permits the patient to quiet down and also gives time for the onset of local anesthesia. Cocainization is fully justified by greater ease of intubation, decreased trauma especially to the cords, and by lessened likelihood of straining during the course of anesthesia even when it is kept on a very light plane. This last point is quite important in brain surgery. In our hands cocaine has proven superior to pontocaine.

We have always preferred the Magill soft-rubber curved catheter for endotracheal anesthesia but it seemed as long ago as 1935 that the orthodox side position of the bevel was a handicap during intubation. Unless the cords are widely separated, the point of this tube usually impinges upon one of them and, with the sharp tips frequently used, may cause definite damage. Therefore we made up two modifications of this tube. In one, the bevel was rotated 90° to place it anteriorly with the tip then falling posteriorly in the midline with the catheter in position for passage into the larynx. This catheter has now been used for five years for transnasal intubations and since the advancing tip falls posteriorly where the cords are most widely separated instead of at the side where one cord may always obstruct tube passage and be somewhat traumatized, this would seem to be an improvement.

The Magill catheter was modified for intubation through the mouth by rotating the bevel 90° in the opposite direction so that in position the beveled side is posterior and the advancing tip is anterior to hook forward easily into a partially exposed larynx.

After cocainization of the larynx, the largest nasal tube which will pass easily without undue force or trauma is well lubricated throughout its length with white vaseline and carefully inserted under direct vision laryngoscopy. About half of these cases will receive the tube without coughing or straining and with no cyanosis. In the rest, immediate connection to the ethylene machine will restore quiet breathing in a few seconds. The tube is carefully fixed to the face against padded gauze with adhesive tape. The position is such that all tube curves are gradual and kinking is unlikely. Several minutes of quiet ethylene anesthesia is allowed before trans-

fer of the patient to the operating table where absolutely proper fixation is of the utmost importance. The gas machine connection to the tracheal catheter is made through a semiflexible rubber tube 12 to 18 inches long and so fixed along the patient's chest that his head may be shifted to any operating position without dislodging or kinking any of the tubing.

While it is perfectly true that a much simpler intubation technic may be satisfactory or desirable in other cases, it is felt that in this group every precaution should be taken against mishap. Here the essentials would seem to be as follows:

(1) All instrumentation must be careful, deliberate, and non-traumatizing.

(2) Every step should be so carefully planned and executed that repetitions will not be necessary.

(3) The final result should allow the lightest anesthesia with the least respiratory obstruction.

(4) Coughing, straining, and cyanosis should not be allowed at any stage.

Repeated efforts at blind intubation, prolonged laryngoscopy under light anesthesia, or injudicious use of ether to deepen anesthesia will all cause marked mucous secretion to interfere with subsequent intubation by any method and later to give some tube obstruction. All of these things should be avoided in the first place.

Final draping of the patient should be such as to allow the anesthetist access to the patient's face where he can check for tube kinking or changed position of the head on its rest.

From this point on anesthesia is conducted on as light a plane as possible with more than adequate amounts of oxygen at all times. This is possible with nitrous oxide without ether, and easily possible with ethylene.

Blood pressure, pulse, and respiratory rates are recorded every fifteen minutes with the exact stage of the operation. This last point is important because of the great bearing it has on the curve changes. We have found respiratory rate to be extremely stable, decreasing slightly only for too deep a stage of anesthesia or a very advanced stage of shock.

Blood pressure drops slightly from tribromethyl alcohol (avertin) anesthesia, tends to rise back to preoperative level during intubation. There is an additional slight rise during incision and another during trephining and sawing out the bone flap. When the bone flap is broken back there is usually an abrupt drop to preoperative level or below and when the dura is opened there is a further drop which is greater when the intradural pressure is greater. Additional drops in pressure may result from the trauma of surgery, as in removing a large deep tumor, or from hemorrhage, and sweating may now become prominent. Needless to say, a fall in pulse pressure is more significant than a lowering of systolic pressure alone.

Pulse rate is little changed, except for slight rises at incision and for trephining, until the dura is opened when it may rise 20-30 points if the intradural pressure has been great. Blood loss and trauma cause additional rises.

Fine judgment is required to decide what treatment to use for falling blood pressure and rising pulse and when to begin it. The rate of change and the amount of deviation from what should obtain for the operative stage are the most important factors. For example, a pressure fall of 10 points during trephining, when it should rise, is more alarming than a pressure fall of 20 points on opening the dura when it should fall anyway. When pressure-pulse changes shift towards shock more rapidly than the operative stage should cause, and some shocking procedure is about to begin, prophylactic measures are in order, but if such changes occur at the end of such a procedure compensation may be automatic.

The first anti-shock measure to take, especially if there is some sweating, is to increase the oxygen percentage in the gas mixture. The second step is to start an intravenous injection of 5 per cent glucose in normal saline solution which is kept ready for immediate use in all of these cases. The rate of this injection should usually be very slow (100 c.c. in 10-15 minutes). If pulse-pressure changes are not slowed or halted or especially if there is active hemorrhage, citrate blood or plasma from the plasma bank is substituted for the glucose solution.

During the last five years increased oxygen has of course been used in the latter stages of practically all cases and glucose has been given intravenously during operation to 30 per cent, but transfusion on the table has been necessary in only 12 per cent of cases.

The average time after beginning of operation for intravenous support to be needed was slightly over an hour and a half but this was because the average total duration of operation was slightly over three hours and the work inside

the dura was usually well under way in one and a half hours.

Oxygen is now given at least during the reacting period to more than half of these cases. Since it allows full nursing care, the nasal catheter method is preferred.

The distribution of operations in this series is shown in the following table:

		Deaths
Craniotomy with decompression	25	1
Cerebral tumor	21	6
Cerebellar exploration	8	4
Fifth nerve resection (intracranial)	7	1
Brain abscess	5	3
Pituitary tumor	5	2
Medulla-cord exploration	4	0
	75	17

Of the 17 deaths recorded, one was on the table of shock and hemorrhage, two died one day later and six died two days after operation. All of these latter cases showed cerebral edema with hyperpyrexia in addition to shock. It is believed that anesthesia was not a major factor in any of these deaths. There was no demonstrated pulmonary complication in any case.

I am glad to have this opportunity to express publicly my appreciation for the privilege of having been able to work with Dr. John J. Shugrue of Washington who performed these operations. Dr. Shugrue and I believe that when the anesthetist exercises good judgment in the general and supportive care of the patient, that the surgeon can do better and faster work. He does not want to be given frequent pulse and blood pressure readings but only to be advised from time to time of the patient's general condition and especially of unusual developments which might dictate a change in operative plan.

It is recognized that such specialized anesthesia as this may have little appeal for many anesthetists. However, there are connotations which have wide applicability. It is possible to see in a few hours in a brain operation the principal physiologic changes which may even require weeks on the ward in a patient progressing from relatively good health to the point of death. The first segments of these curves will resemble those seen in good risk general surgical patients coming up for short operations; the central segments may simulate the curves of fair risk patients undergoing more extensive surgery; while the terminal segments of these curves will be similar to those of bad risk patients under serious surgery. Thus the teaching value of these brain cases is great even in a relatively small series. They merit the careful study of any anesthetist.

SUMMARY

(1) For brain surgery, general anesthesia causes less strain on the surgeon and patient.

(2) The recommended routine calls for the use of tribromethyl alcohol (avertin) basal anesthesia followed by endotracheal ethylene (or nitrous oxide when the electrosurgical unit is used). The gas is given in a closed system with carbon dioxide absorption.

(3) Direct vision laryngoscopy is used for cocainization of the larynx and for intubation.

(4) A modification of Magill's tracheal catheter is described.

(5) Fine judgment is required on the part of the anesthetist in general support of the patient.

(6) Attention is called to the need for meticulous care and deliberation in all steps of this work.

(7) The teaching value of this group of cases in relation to the general surgical group is outlined.

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DISCUSSION (*Abstract*)

Dr. Exum Walker, Atlanta, Ga.—The primary effect of nitrous oxide is anesthesia by exclusion of oxygen, anoxemia if you will. So many of our patients have to be anesthetized over very long periods of time and are very poor risks to start with, that if they are anesthetized by anoxemia, it is a cause of fear and trepidation to me. If I am incorrect about the physiologic action of this drug, I apologize, but from a practical standpoint I have never seen it used very satisfactorily.

As to ethylene and cyclopropane, the chief disadvantage is that they are highly explosive.

I am one of the neurosurgeons who has come to use the electrocautery almost constantly in my operative procedures, and I do not know how I could get along without it. Some of the hospitals have gone so far as to forbid me to use the cautery with cyclopropane or ethylene, and for that reason I have looked eagerly for some other method. When the above drugs have been given, in my experience, there has always been some increase in bleeding, although not quite so much as with ether, it is true. Ether causes increased intracranial pressure, increased bleeding all the way through, which from the technical standpoint is our primary problem.

Most neurosurgeons try one anesthetic agent and then another and ultimately come back to procaine. Procaine is not ideal for several reasons. One, it takes time for the surgeon to stop and infiltrate the field of operation. It is quite an ordeal for the patient to go through a long procedure being conscious of what is going on, particularly of side remarks in the operating room.

Third, there is the drop in blood pressure. I have used procaine many, many times, and I have been struck by the number of times that I have gotten a drop in blood pressure following its injection, and just before I began my incision.

As to tribromethyl alcohol, I have been an enthusiast, have used it quite a good deal, and think it is a wonderful drug. It has some disadvantages in that the individual who gives it must be thoroughly familiar with it. It is time-consuming before the operation. The individual dose for the patient cannot be accurately determined. If you estimate the dose, you do not know how much of it is going to be absorbed. That depends upon the variability of absorption from the rectum, how much surface mucosa it comes in contact with, and also how dehydrated the patient may be. If the patient has an overabundance of fluids, absorption will be very slow. If he is very dehydrated, absorption will be rapid. That, of course, is the reason that tribromethyl alcohol has always been used as a basal anesthetic. When it is used in conjunction with something else, I have no criticism at all of tribromethyl alcohol.

Six or eight years ago I had in my mind's eye a fantastic picture of the day when we would stick a needle in a vein and administer an anesthetic agent that would act, instantly, and be quickly excreted. I hoped that at the same time we could utilize the vein for putting in fluids, perhaps drugs, artificially to control blood pressure and more or less to take over the basic vital functions of the patient. This dream has not come true, but we have made considerable progress.

When sodium ethyl thiobarbiturate began to be used over the country, it attracted my eye. In some hospitals it was given more or less rapidly, perhaps while the patient was counting. When the patient stopped counting the surgeon began the operation, and if it was not finished in time, an additional dose of the drug was given. I noticed that the patients often became ashen and cyanotic, and when the skin of the ear or forehead was compressed took considerable time for the circulation to recover. I presume that means some paralysis of the capillary bed. For that reason this may not be a safe anesthesia, thinking primarily of the circulation of the brain stem, and the permanent damage to the brain which might occur from such an anesthetic. Incidentally, I also noticed that many of these patients slept for a long time after operation.

Finally, when we began using sodium ethyl thiobarbiturate ourselves, we used a different method of administration. Perhaps it is employed in other parts of the country. I do not know.

We give it, drop by drop, allowing five to ten minutes for the patient to become anesthetized. The anesthetist sticks the patient with a pin, and when the time comes that the patient does not respond, the operation begins.

After I have made my skin incision, if the patient moves a little, the anesthetist gives him another drop or two, and it may be two or three or five minutes before I continue. We give just enough drug for a minimal anesthesia. In intracranial work, after the skin incision has been made, there is very little pain. We do not disturb the patient very much, so he can be carried very lightly.

As the procedure goes along, if the patient begins to move a little, we give a little more ethyl thiobarbiturate, slowly, drop by drop. We have used a two-way stopcock for the needle, the anesthetic going in one side, and the saline in the other. Saline is allowed to run in very slowly at first because we found out if it goes in rapidly bleeding is increased.

The blood pressure is taken every three minutes. Five or ten per cent glucose may be added to the saline so if we begin to get a little drop in blood pressure we artificially control it. Having our infusion already started, we do away with the necessity of finding an intern and spending an hour trying to get some fluid started with the blood pressure low.

We have to date I believe used sodium ethyl thiobarbiturate in 111 cases. We have not had a single death attributable to the anesthetic or the operation. The earliest death we have had has been two and a half weeks postoperatively, so I do not believe you can blame the anesthetic for it.

The longest period we have carried a patient under the influence of this drug has been eight and a half hours.

About ten days ago we had a woman 72 years old with a blood pressure of 220/130. We carried her seven hours, and she was talking to us ten minutes after she left the operating room.

We have given fluids through the operation so that they get anywhere from 1500 to 3000 c.c. We give fluids as slowly as possible during the main course of the operative procedure, but afterwards we see that they get additional fluids because we think fluids increase the excretion of the anesthetic, and I believe it has recently been pointed out that glucose helps decrease its toxicity. Almost all our patients have been able to talk within a half hour afterwards and practically all of them respond to painful stimulation when they leave the operating table.

I have tried to cooperate to the utmost with the anesthetist. I always ask him to see the patient beforehand, and leave the ordering of the preliminary drugs and the whole anesthetic problem to him. It relieves me of much of my responsibility. We frequently get together and discuss any points or ideas we may have, trying to help each other.

These few cases do not necessarily indicate that this is a safe drug, but we believe its advantages justify its risks. From a practical standpoint, it has been a Godsend. The patients' blood pressures have hardly varied; we have not had any outstanding drops. The patients are perfectly quiet, their breathing is quiet, and for that reason bleeding is less. The bleeding has been comparable to bleeding in spinal anesthesia or in local anesthesia.

Dr. Joseph B. Bogan, Washington, D. C.—The hospital in which I work has had but little neurosurgery of late years, and there has been a tendency on the part of the surgeons definitely to request tribromethyl alcohol for use with procaine infiltration.

I rather object to a method which does not present the anesthetist with an opportunity properly to control respiration, and I think a combination of tribromethyl alcohol and procaine infiltration is such a method.

Ordinarily, however, in spite of the fact that the operation has been scheduled to be done under procaine plus tribromethyl alcohol, the eventual outcome is that the patient is given a little nitrous oxide oxygen intratracheally.

Dr. Stubbs (closing).—When the use of nitrous oxide is attended by any degree of anoxia or respiratory obstruction, hemorrhage is worse and shock more likely to occur. Oxygen must be not less than 20 per cent of the respired gases and an endotracheal tube clear of mucus, blood, or other obstruction must be used to avoid these difficulties. Even the weak anesthetic value of nitrous oxide is enough to furnish the light anesthesia needed for these cases, even when surplus amounts of oxygen are given.

The combination of basal with gas anesthesia has worked for us. It is perfectly true that the use of tribromethyl alcohol requires considerable care on the part of the anesthetist, more than is frequently given. It is not merely a matter of pouring it into the rectum and leaving it to take care of itself. The main reason we prefer to use a gas in conjunction with tribromethyl alcohol is that we then have both anesthetic and respiratory control.

I should like to agree also with the idea that early intravenous fluids are somewhat contraindicated; that is, contraindicated unless there is a demonstrated need for them. Since we are trying to maintain a level of circulation rather than to get above it, we can do definite harm by injecting fluids too early.

I believe the four major points in our routine should be individually evaluated as follows:

Most important is an endotracheal airway which I believe should be used for all of these patients under general anesthesia whether the agent be gas, ether, ethyl thiobarbiturate, or any other, because it insures control of respiration in all operative positions and for any emergency.

Since an endotracheal technic is desirable, supplemental gas (ethylene or nitrous oxide) gives the best and safest anesthetic control, and is therefore listed second.

Next in importance is use of the carbon dioxide absorption technic which preserves warmth and moisture for the patient and promotes quiet, easy breathing.

Least important is the specific use of tribromethyl alcohol as a basal anesthetic. However, something approaching basal medication aids greatly in smoothing out a long anesthesia.

We have a general surgical service with only a few neurosurgical operative cases. We felt that it was worth while to study them as thoroughly as possible because of the bearing they have on the rest of our surgical cases, and because of the feeling that just because we had only a few cases of a specialized type we should not be excused for doing a poor job in handling them.

NEPHRITIS*

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THE NORMAL CAPILLARY AND THE PREVENTION OF EDEMA

The tissues of the body are nourished by solutions of certain essential substances which pass from the blood through the walls of the capillaries into the intercellular tissue spaces. These substances will be referred to herein as salts.

The walls of normal capillaries allow the water of the blood and these salts to pass through them in both directions, but they do not permit the

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passage of proteins. When capillaries are damaged the albumins and globulins also pass through their walls and gain access to the intercellular tissue spaces.

The force that we refer to as the blood pressure drives the salt solutions out of the capillaries into the tissue spaces. At the arterial end of the capillary the blood pressure is equal to 32 millimeters of mercury. This degree of pressure is practically a constant figure and does not vary with the height of the blood pressure in the main arterial system. Whether an individual has a systolic blood pressure of 200 mm. or 120 mm. the blood pressure at the proximal or arterial end of the capillary is the same, that is, 32 mm. When the blood reaches the distal or venous end of the capillary the pressure normally has dropped from 32 to 12 mm., a difference of 20 mm. At the mid point of the capillary the pressure is one-half the difference between the initial and final capillary pressures, or 22 mm.

This force which tends to expel the salt solutions from the capillaries is opposed by the osmotic attraction of the albumins and globulins of the blood. The average value of this osmotic attraction of the blood proteins is equal to 22 mm. of mercury and therefore equals the hydrostatic pressure at the mid point of the capillary. At the mid point of the capillary, the blood pressure which tends to drive the salt solutions out is counteracted exactly by the colloidal osmotic attraction of the proteins which tends to draw them back.

From the arterial end of the capillary to its mid point the blood pressure is higher than the colloidal osmotic pressure; therefore the predominating movement of the salts and water is outward. Whereas, from the mid point of the capillary to its venous end the blood pressure is lower than the colloidal osmotic pressure; therefore the predominating direction of the flow is inward. Given a normal capillary, the amount of fluid and salts forced out along its proximal half by the blood pressure will be equaled by the amount drawn back into it along its distal half by the colloidal attraction.

This is the normal course of events and the constancy and equivalence of these opposing forces prevents water-logging of the tissues on one hand and dehydration on the other in spite of the great variations in the intake and output of fluids that occur from day to day and from hour to hour.

THE DAMAGED CAPILLARY AND EDEMA FORMATION

In nephritis with edema the precise balance of these opposing forces is disturbed. First in acute nephritis an increase in the permeability of the capillary walls occurs as a result of injury to them by bacterial toxins in the blood. This increased permeability allows a greater than normal quantity of water and salts to pass into the tissue spaces from the blood stream. But of even greater importance it also allows some of the albumins and globulins of the blood to extravasate into the surrounding tissue spaces. Such a situation gives rise to the initial edema which we see following severe infections and which we recognize as the forerunner of or the actual beginning of acute nephritis.

This damage to the capillaries with resulting increased permeability of their walls leads to a disparity between the finely balanced forces which preserve the equilibrium of fluid exchange between the blood and the tissue spaces. For when the proteins of the blood plasma gain access to the tissue spaces outside the capillaries, they exert osmotic attraction in the wrong direction and aggravate the edema which is already present by opposing the osmotic attraction exerted by the colloids still remaining within the capillaries.

THE PLASMA PROTEIN DEFICIT AND EDEMA FORMATION

Another factor in edema formation now comes into play, for if we assume that the initial damaging effect of bacterial toxins on the capillary walls has ceased and that the capillaries have become restored to their normal permeability for salts and water only and not for proteins, it must be borne in mind that the proteins remaining in the blood have been reduced in amount and can therefore no longer exert their normal attractive force. The result is that the point of equilibrium between the blood pressure which forces solutions outward and the colloidal osmotic attraction which draws them back is no longer situated at the mid point of the length of the capillary, but has become shifted toward its venous end. Consequently the salt solutions are forced out of the capillaries for a greater portion of their length than that during which they are drawn back. This results in more salt and fluid's leaving the blood stream than returns to it and edema becomes progressively worse.

SODIUM CHLORIDE AND EDEMA FORMATION

Theoretically the only limit to the degree of edema that can result from this mechanism is

the distensibility of the skin, for if the skin becomes taut from over-distention its pressure on the contained tissues would counterbalance the force which is tending to drive more fluid into the tissue spaces. In the loose areolar tissues covered with elastic skin such as the eyelids and scrotum we see marked degrees of edema. Actually the amount of edema that occurs is governed by the amount of edema-forming material that the patient is taking in. This refers to sodium chloride and water and will be given proper consideration presently.

ACUTE NEPHRITIS

Leaving these considerations for the moment and turning our attention to the clinical aspects of nephritis, we are confronted at once with a multiplicity of classifications, those of Osler, Widal and Maclean, Volhard and Fahr and Christian being the most familiar. All classifications, whatever their terminology, must recognize four outstanding syndromes: acute nephritis, chronic nephritis with edema, chronic interstitial nephritis and arteriosclerotic nephritis.

The first of these, acute nephritis, usually occurs during or toward the end of an infectious disease such as scarlet fever or other streptococcus infections. Fever, headache, backache, nausea and anorexia are frequently present. It is characterized by edema, which is apparent first in the loose areolar tissue about the eyes, but later is seen to involve the entire body. The urine is scanty and contains albumin, blood and casts. There is but little elevation of the blood pressure and no nitrogen retention.

The treatment of this condition consists of rest in bed and the eradication of the infection whose toxin is primarily responsible for the injury to the capillaries, and the neutralization of this toxin if possible with the proper antitoxic serum. As this condition is usually of relatively short duration, certain liberties may be taken with the patient's nutrition. Fruit juices, milk and water with added glucose may be given for several days. After this a salt-poor diet should be given and increased cautiously if all goes well until a full nutritious diet is being taken.

During this period ample opportunity is presented for making the laboratory examinations necessary to appraise the severity of the injury. It is desirable to examine several specimens of urine, perform renal function tests, keep accurate account of the intake and output of fluid and ascertain the level of the nitrogenous ingredients and the sodium chloride of the blood.

Should the patient's condition become unsatisfactory with diminishing output of urine various measures should be instituted to promote diuresis such as increase of the fluid intake, hot packs, glucose solution intravenously and the application of continuous negative pressure to a retention catheter in the patient's bladder. The latter measure has served well on several occasions when all other measures to relieve anuria had failed.

The prognosis in this type of nephritis is good, but rest in bed and strict adherence to dietary regulations must be insisted upon until all clinical and laboratory evidence of the disease have disappeared.

CHRONIC NEPHRITIS WITH EDEMA

The second type of nephritis with edema is known under various designations: chronic parenchymatous nephritis (Osler), nephritic type of glomerular nephritis (Volhard and Fahr), chronic active stage of hemorrhagic nephritis (Addis), and so on. It is a chronic condition of insidious onset characterized by edema and by urine containing a large quantity of albumin and a varying quantity of blood. In this type there is no important elevation of blood pressure and no inability on the part of the kidneys to excrete nitrogenous waste products. Many authors are of the opinion that it is always a sequel to an attack of acute nephritis which in some instances was so mild and transitory as to have escaped notice.

It is in this type of nephritis that the proteins of the blood plasma are reduced in quantity due partly to the initial extravasation of albumin and globulin through the capillary walls into the tissue spaces and partly to the continuing loss of albumin in the urine.

Within the memory of many of us, protein restriction was a generally employed therapeutic measure in all types of Bright's disease. Such restriction arose as a logical deduction from the facts that protein metabolism produced urea, that in Bright's disease the ability of the kidneys to excrete urea was impaired and hence that the work required of the kidneys could be made lighter by reducing the protein intake.

HIGH PROTEIN DIET

In view of the modern concept of edema formation, it appears that patients with this type of nephritis require, for the regeneration of

plasma proteins and for the maintenance of nitrogen equilibrium and the normal mass of body tissue, diets containing more protein than would keep a normal individual fit. In these patients the chief incapacitating factor is the edema connected with the plasma albumin deficit. Their most urgent need is a sufficient increase in their plasma albumin to bring the edema under control. This need can be met by a diet containing approximately 100 grams of protein per day; that is, for an average man about 1 gram per kilogram of body weight or 60 to 70 grams to replace the normal wear and tear on the tissues plus 15 to 20 grams to replace the albumin lost in the urine and 15 to 20 grams more to build up the wasted tissues which are uniformly encountered and which become painfully evident after the edema has subsided. It is essential to give sufficient carbohydrate and fat to meet fully the caloric requirements of the patient so that he may use the protein as economically as possible.

SODIUM CHLORIDE RESTRICTION

There is another controllable factor in edema formation besides the level of the plasma proteins. This is the sodium chloride consumption. While a deficit in the plasma proteins is the most important condition favoring the production of nephritic edema, it must be borne in mind that edema cannot occur if the materials necessary for its formation are not supplied. These materials consist of sodium chloride and water in the proper proportions to form physiologic saline solution. Retention of water does not occur without sufficient sodium chloride to form a solution approaching isotonicity with normal body fluids. So if a potentially edematous patient takes sufficient water to prevent thirst the extent of the edema formed is largely proportional to the sodium chloride content of the diet.

Experimental and clinical data exonerate the kidneys themselves of any direct responsibility for edema. The kidneys of edematous patients can excrete water in the normal fashion when it is presented to them and these kidneys are also able to excrete the normal amount of waste products, including sodium chloride. In short, edema is not due to the inability of the kidneys to excrete water, sodium or chlorides. This concept of edema has fostered a new and encouraging type of therapy. It endeavors to move sodium from the tissues into the blood stream and it insists upon an intake of water sufficient to compensate for the impaired ability of the kidneys to form concentrated urine.

It must be stated as an unsatisfactorily explained fact that while there is a tendency to retain physiologic sodium chloride solution the organism refuses to retain salt solutions which deviate in mineral content from the chemical composition of the intercellular tissue fluids. In other words, potassium chloride may be supplied to an edematous patient with impunity. A teaspoonful a day will be a fairly satisfactory substitute for common salt as seasoning for his otherwise salt-poor food.

ACID SALTS AND WATER AS DIURETICS

Acid or acid-producing salts will liberate sodium from the tissues. Ammonium chloride or ammonium nitrate may be used for this purpose. As they are gastric irritants, they should be administered as 0.5 gram enteric coated tablets in doses of 3 to 10 grams daily. The flow of sodium chloride from the tissues into the blood presents a large additional amount of waste material to be excreted by the kidneys. Since it may require as much as 50 c. c. of water to remove each gram of salt, it is essential that the available water be sufficient for that purpose. Adequacy is assured as long as the specific gravity of the urine is below the maximum that the kidneys can attain.

PROTEIN SUBSTITUTES INTRAVENOUSLY

If these measures all fail to institute free diuresis, there yet remains a therapeutic measure based upon the mechanism of edema formation described above. The colloid osmotic pressure of the blood may be increased by intravenous injections of natural blood proteins as by transfusions either of blood plasma or blood serum. Or artificial substitutes for serum proteins may be used. The intravenous injection of hydrolyzed casein solutions to which the amino acids tryptophan and cystine have been added is still in the experimental stage. Gum acacia is at present the most popular. Acacia solution is obtainable in ampoules of 100 c. c. of 30 per cent solution. This is diluted with distilled water to 500 c. c. so as to make a 6 per cent solution for intravenous injection. This may be repeated and frequently disposes of an otherwise insoluble situation.

CHRONIC INTERSTITIAL NEPHRITIS

Of the third and fourth types of kidney disease dealt with in this paper but little will be

said. The third type is known as chronic interstitial nephritis (Osler), azotemic nephritis (Widal-Maclean), vascular interstitial glomerular nephritis (Volhard and Fahr), and latent and terminal stages of hemorrhagic nephritis (Addis). In these cases the albumin excretion in the urine is negligible in quantity, there is a fixation of the specific gravity in the neighborhood of 1.012, high blood pressure and retention of nitrogenous products, but no edema. In the terminal stage of this type the toxic manifestations known as uremia are likely to occur. Since the kidneys require a large amount of water for the excretion of waste products because of their inability to elaborate concentrated urine the treatment calls for a large intake of fluid and a minimal amount of waste products to be excreted.

This statement needs some explanation and elaboration. It will be recalled that a simple method of calculating the total solids in a liter of urine is to take the last two figures of the specific gravity and multiply them by 2.33 (this is known as Haeser's coefficient). For example, if the specific gravity of the urine is 1.020 we take the last two figures 20 and multiply by 2.33, which gives 46.6 grams as the total solids in one liter of urine. If the urine has a specific gravity of 1.010, the last two figures, that is, 10, multiplied by 2.33 will give 23.3 grams per liter of urine. If a patient has 46.6 grams of total solids to eliminate from his system per day and excretes only one liter of urine having a specific gravity of 1.010, it is evident that he will excrete only 23.3 grams of solids, leaving another 23.3 grams to accumulate in his blood and tissues. In order to excrete this additional 23.3 grams of waste material, he must take in enough water to excrete another liter of urine having a specific gravity of 1.010. If one patient excretes one liter of urine having a specific gravity of 1.020 and another patient excretes two liters of urine having a specific gravity of 1.010, both will excrete 46.6 grams of total solids. So if a patient's kidneys can elaborate urine the highest specific gravity of which is 1.010, he must obviously be given sufficient water so that he will excrete two or more liters of urine. This is the basis for giving large quantities of water in this type of nephritis.

ARTERIOSCLEROTIC NEPHRITIS

The fourth and last type is arteriosclerotic nephritis or nephrosclerosis. The clinical picture is quite like the previous type, but is usually attended by cardiovascular accidents as cerebral hemorrhage, coronary sclerosis and occlusion or heart failure. The general vascular system is primarily at fault and the functions of the kidneys are affected secondarily. In this type also there is a tendency to edema formation, but here the edema is of the dependent or cardiac type. Relative failure of the right heart leads to increased venous pressure so that the drop in blood pressure along the capillary from the arterial end to the venous end may be only 14 mm. instead of the usual 20 mm. As the colloidal osmotic pressure is not altered the point along the capillary where the hydrostatic pressure and the colloidal osmotic pressure are equivalent is shifted toward the venous end. Hence more fluids are driven out of the capillaries by the blood pressure than are drawn back into them by the colloidal osmotic pressure. Here in addition to the usual attention to protein, salt and water intake, digitalis or one of its close pharmacologic relatives will be administered to full effect.

FUNDAMENTALS OF TREATMENT OF THE PSYCHONEUROSES

By HORACE K. RICHARDSON, M.D.
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Unfortunately the treatment of the psychoneuroses has not yet reached that stage of precision, and is not so idiomatic, as is that in certain of the specific biochemical or infectious disorders; therefore, in the hands of the psychotherapist, the treatment of these troublesome conditions is very largely determined by, and is dependent upon, the particular theories, disciplines and experiences which go to make up the patterns of his own acceptances and convictions. As is well known, these vary considerably among different individuals and in various groups. Essentially, however, psychotherapy tends to orient itself and to crystallize around the more or less theoretical assumptions of the five or six contemporary so-called "schools" of psychological thought, and according to their psychopathological postulates and technical methods of investigation.

One therapist may approach the treatment of his psychoneurotic patient from the viewpoint of the introspective or existential school; another

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may be so influenced by the Gestalt ideologies that he will attempt to explain departures from the normal through theories developed within this group, or he may follow Watson and the behaviorists and apply the theories of the condition reflex to the solution of his psychiatric problems. He may be a disciple of Sigmund Freud or his various pupils; Jung, Adler, Stekel or Rank, and utilize the theories and technic of psychoanalysis exclusively, or modified, for the basis of explanation and insight into the expressions of psychopathology. He may go along with McDougal according to the purposive or hormonic school, or with Adolf Meyer and the psychobiological concepts, or he may take the middle of the road and become an empiricist.

Regardless of the "school" or pattern of thought which he follows, or the various technical methods which he may use, the therapist is ultimately confronted with the practical realization that he is dealing with a human being who is in trouble, and who has come to him for assistance in his attempt to find a solution to a problem which has been, up to this time, well beyond his capacity to solve without help. Regardless of which set of theories he will use, one great fact will stand out in his mind, namely, that he is confronted with two essential factors which must be looked upon as the material with which he will be obliged to work: first, that there is a situation; and second, that there is a personality involved in the situation. It is these two fundamental factors that must be investigated, and one or both of them must be modified. Naturally, if the first only is changed, the personality will remain essentially unchanged and therefore subject to further maladaptations. It will be discovered, generally, that the first of these two factors provides the precipitating causes for the break in maladaptation, and that it is the second factor, namely, the personality, which is the more responsible for the difficulty and which will receive the major part of the attention in the therapeutic effort. Because of the limitations in time, it is this factor only, the personality, which will be discussed.

Obviously the facts of the situation must be obtained. They will consist of the history, personal and familial, the results of physical and laboratory examinations, a description of the environment, and the individuals who go to make it up, a statement of the complaint, and so on. With the disclosure of as many relevant facts as may be obtained, the therapist is ready to proceed to the second factor, the personality. Upon the results of this part of the investigation, the ultimate therapeutic result will be largely determined.

Now how shall we proceed to investigate this important factor, the personality? Personally, I believe that we can make little mistake if we hew as closely as possible to the line of biological and neurological development, within the limitations of the knowledge we have at hand. I should not discard entirely any of the theoretical assumptions of any of the various schools. On the contrary, I should attempt to know and use as much as possible of all of them, and to keep them in mind continually with the purpose of seeing how any of them may square with the facts as they are developed. I should not put all my faith in any one exclusively, however. While the approach to a study of the personality through the more or less hypothetical avenues of certain schools of psychological theory frequently offers intriguing and startling explanations for human conduct, I believe that the biological and neurological developmental avenue, and the concepts which are correlated with this approach, furnishes the most convincing foundation upon which to erect a successful curative system of effort, one which will stand the test of practical and continued experience. I should like to state at this point that nothing that I may say, or neglect to say, minimizes in importance, or denies in my own thinking, the presence of unaware or forgotten, or "unconscious" if you will, determinants and influences which tend to shape and determine patterns of behavior on all the various levels of reaction. In fact, without the acceptance of the possibilities of such influence, human behavior becomes almost inexplicable. Neither may we safely neglect this source of material in the search for the origins of the maladaptive expressions.

As has been previously stated by the writer,[1] we must presume, at the outset, the acceptance of the principle of psychobiological integration in contradistinction to the tenent of parallelism. Body and mind are complements of each other, not supplements to each other. We must accept the fact that there is a relationship between the various physical and psychic levels. As has been said, "Can anyone imagine function without structure?" This implication includes a unity of action even more generally apparent than our present knowledge of the structural systems discloses. This interdependence and synthesis of function transcend our knowledge of nervous system localization. We must look upon the troubled mind, the maladapted person,

not as a group of physiological and psychological fragments, but as an integrated, functioning whole, which is attempting to adapt and adjust itself to life. The very ideals of life and of mental health are based upon this unity of the personality. As Dr. Frederick Tilney has said,

"In the main we have made disease our chief interest. As a matter of fact, life is the principle theme, to which disease is but a corollary. The purely pragmatic attitude toward medicine is not without its defects. It fails to encourage an approach to the salient problems concerning the significance of life. It omits, as theoretical, the considerations of development and adaptation."

In our therapeutic program, this ideal becomes the basis for understanding, not only of integration and the lack of it, but of disintegration as well. Until this fact is understood and accepted by the patient, a valuable adjunct to a final synthesis of the personality is lost to him. I believe, therefore, that the development of a working knowledge of this concept upon the part of the patient constitutes one of the first steps in the process of therapeutic re-education. A review of the factors behind this principle is the next logical step in our program.

We believe that each individual personality is made up of the end results of development, the residuals of growth and experience, both phylogenetic and ontogenetic, integrated into certain actual and potential constituents which express themselves in four more or less predictable reaction types: the reflex, the instinctive, the intelligent, and the ideal. These reactions are apparently based upon the four developmental levels of the nervous system. They therefore explain the purely biological foundation of the concept. From the functional aspects these four neurologic levels are repeated in the growth of the individual from birth to maturity, and therefore provide a logical basis of understanding for both the mature and the immature expressions of personality. They explain the phenomena of retardation, fixation, disintegration and the antecedents of individuality and character; and when considered in the light of individual hypersensitiveness, they explain the degree of all dynamic responses. It is recognized that in this statement there is much that is controversial in nature. The concept is presented to the patient, however, simply as a postulate upon which to erect a working degree of understanding and explanation. It has been found, by practical experience, to be understood easily by the patient, acceptable to him, and useful as a foundation for the objective use of the personality as an integrated whole.

The first of these developmental levels, the reflex, represents the lowest type of biological adaptation as implied in the functions of the primitive brain stem. It implies certain primitive, non-affective responses which occur outside of the awareness, and which, by the conditioning process, have become incorporated into more or less automatic, neurologically determined functions commonly known as habits. This primitive structure is represented in general by the spinal cord and the medulla. An understanding of the functions and individual expression of this level of habit reaction, upon the part of the patient, is one of the first steps in the therapeutic excursion.

The second type of reaction is due to the development of the next higher level of neurologic structure, the diencephalon. As the thalamic region of the brain developed, there arose another type of reaction, known as the instinctive. This is not a simple mechanical reflex, but it involves the entire psycho-physical organization, as has been ably demonstrated by Cannon, Alvarez, Dunbar, *et al.*

These instinctive reactions seem to be essentially associated with the protective needs of the individual, both as an individual and as a member of the race as a whole. They involve the various physiologic structures and functions associated with the autonomic nervous system. In other words, this neurologic level of reaction appears to have as its main function the preparation of the individual, both physically and mentally, for the greater total body effort required by certain types of situations arising both within the self and within the environment. It seems to be especially associated with the mechanisms of escape and protection, originally for the physical being and, in later developmental periods, for the ego and total self.

The development of insight, upon the part of the patient, into the nature of these instinctive reactions, and especially into those which have contributed largely to the origin of his own conflicts, is the second logical step in the growth of therapeutic insight.

The third type of reaction is the intelligent. This came about through further development of the telencephalon, or end-brain. With growth in this part of the nervous system, came new capacities and abilities, a new function, namely, thinking.

"Just what intelligence is, we do not know. Let us limit its meaning for our present use to its functional significance."[1]

It is the ability to comprehend, to form voluntary decisions and judgments, but principally to use the power of choice. It is this faculty which constitutes man's ability to make the adaptation purposive, and it is this particular level of reaction which man must learn to use to a greater and increasing degree if he expects a new orientation to life.

Finally, there comes the fourth or highest level of reaction as represented neurologically by the growth and development of the neopallium. This level, functionally, is known as the ideal or ethical level; and it is here that man's most difficult type of adaptation takes place. It is because of this level that man finds himself in conflict, not only with himself, but with the elements of his environment.

As man found it desirable to live in groups, it became necessary for him to modify the gratification of his instinctive desires and demands in order that the good of the group as a whole should take precedence over what appeared desirable and satisfying to the individual alone, and through the development of this reaction, as sense of individual responsibility to the group took form. It is that reaction which is responsible for man's progress toward civilization. It is upon this level that the unified energies of man come to fruition in the form of purposive striving, and it is here that man erects his philosophy of life.

Insight into the part that the ethical level plays in the normal, satisfying, efficient life, its functions, as well as its role in psychoneurotic maladaptation is one of the most necessary concerns of the psychotherapeutic program.

To recapitulate briefly according to the postulate, the various parts of the personality and their respective functional and neurologic implications are: first, the level of reflex under the domination of the medulla with reactions known as habits; second, the archaic, inherent force called instinctive, represented in the neurologic structure by the thalamic region and furnishing the dynamics for the affective or emotional life of the individual; third, the intelligent level, represented neurologically by the fore-brain and providing the function of thinking, with the power of choice as its principal expression; fourth, the ethical or ideal level, represented by the more recent developments of the fore-brain or neopallium, and expressed as a super-intelligence and conscience.

To quote Dr. Austen Fox Riggs, with whom the writer prepared a paper on this same material:

"These are the variables in the human equation which, in their reactions to one another and through their organization as a whole, constitute the bio-psychological entity, the individual, who, in ways that are characteristic of the race, on the one hand, and of himself individually on the other, reacts to his environment and to which the environment itself reacts."[1]

"At birth this instrument is imperfectly developed and immature, and functions only in part, and at its lower levels. It then goes through a gradual process of maturation, determined and affected largely by the nature and character of the environmental influences, pressures, disciplines and authorities which surround it. In short, on its way toward maturity, it may go through certain events, real or imagined, which tend to condition it and perhaps fix its reactions at certain definite chronological levels, and hold them in that part of the mental life which is not at all times in the region of the awareness,"[2]

and which by the laws of association, may become reactivated and be the determinants of conduct, which is inexplicable and confusing to the patient. It is desirable that insight into the nature and meaning of these influences should be developed in the mind of the patient.

The essential basis for the difficulties in the psychoneurotic is believed to be a lack of integration of these personality elements and an accentuated suggestibility and hypersensitiveness to the domination of any one or more of these four various levels of reaction specifically expressed by subsequent conflicts between them, especially between the lower two and the higher two.

The first and essential objective in the therapeutic effort is to develop an understanding of this personality structure and its various individual reactions, and then to ascertain the origins of the basic causes for the conflicts within it. This is accomplished by an analysis of the life situations of the individual, as well as by bringing out all the known factors relative to personality expression which go to make up his disposition, temperament and total character. Finally the effort is made to develop within the mind of the patient an interpretive insight into the origins and nature and function of the symptom picture and its relationship to the maladaptation, and to his real needs. He is then in a position with the help of the physician and his own insight to plan and ultimately to develop adequate solutions to his conflicts by a more skillful and efficient use of these four types of reactions which constitute his total personality. This is the basis upon which the therapeutic re-educational synthesis is evolved.

To sum up this very brief discussion of treat-

ment of the psychoneuroses, I consider the important factor is to give the patient a workable degree of insight into his life habits, his emotional reactions, his methods of thinking and just what part purpose, conscience, and ideals are playing in his conduct of life, and, what is of greater importance, insight into the nature of the influences which brought about the habitual use of these elements of his personality in such a way as to cause his maladaptation. Naturally, there will be many technical methods used in accomplishing the objective of final synthesis and integration, such as isolation, discussion, suggestion, free association, catharsis, explanation, analysis, improving physical status, and many others.

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DISCUSSION (*Abstract*)

Dr. Titus H. Harris, Galveston, Tex.—As Dr. Richardson has pointed out, there are several schools of thought regarding the interpretation of the mechanisms underlying the psychoneuroses, and it is best to utilize what seems practical from every school and apply it in the individual approach to the patient.

Psychiatry has been regarded for many years by the medical profession as being too theoretical and impractical and this perhaps accounts for the lack of interest on the part of the medical profession in it. Teachers of psychiatry in recent years have stressed the importance of utilizing a common sense point of view in dealing with the psychoneuroses.

All psychiatrists accept the principle that in attempting to help a psychoneurotic patient we are dealing with two sets of factors, an individual with his particular equipment and the environment. Dr. Richardson has limited his discussion to means of modifying the personality; not that he minimizes the part played by the situation to which the patient is attempting to adjust.

Teachers of psychiatry are trying to bring about a closer relationship between psychiatry and medicine, and are constantly seeking means of simplifying therapeutic methods, so that every physician can treat his patients from a psychiatric point of view and manage his own psychoneuroses, leaving to the specialist in psychiatry only the more difficult ones. To do this we must find a method of approach which is practical and not too time-consuming. Perhaps for this particular purpose more stress can be placed on the situational factors.

Unquestionably, many of the mild psychoneuroses can be managed quite well by altering the situation and giving the patient a new point of view, which can be done in a relatively short time.

The point of view expressed by Dr. Richardson would lend itself to a more simplified approach, which we greatly desire.

Dr. Martha W. MacDonald, New Orleans, La.—Dr. Richardson has given us a very interesting and provocative paper on the structure of the personality in terms of neurologic levels of functioning rather than a paper on fundamentals of treatment of the psychoneuroses. Few clinical students of the neuroses would disagree with him in his formulation of levels of functioning into reflex, instinctive, intellectual and ideal, although they may have reservations in linking these four levels to structural levels of the nervous system.

As for Dr. Richardson's discussion of levels of functioning, I fail to see wherein this concept adds anything new to our understanding of either personality structure or functioning. Lower and higher levels of functioning have been lucidly described and repeatedly discussed in great detail over a period of some thirty years by a man who was a neurologist as well as an intuitive clinician. He based his theory on painstaking observation aided by that rare quality of emotional understanding and intuition upon how fear, hate and love affect a particular patient. Undoubtedly this intuition was founded upon his heretofore unknown honesty in self-examination, which, in turn, finally led to his formulation of the unconscious. Ever the student, he studied and described the steps by which the individual found defenses at various levels for protecting himself from anxiety. From a study of the defenses he theorized as to why these levels failed to be integrated, why one level assumed dominance, and what might happen when ambivalence and conflict occurred between lower and higher levels, or different drives within the same level. He has carefully stressed and clinically demonstrated the need for altering and re-educating the higher levels so that they in turn may alter and control the direction if not the strength of the primitive instinctual levels.

As a great teacher he recognized that these levels of expression could be altered or redirected through the patient's achieving insight, insight at the deeper levels, emotional insight rather than intellectual insight. This differentiation is frequently overlooked, and I regret that it has not been stressed today lest we think we have discharged our responsibility to our patient when we have made an interpretation which may be acceptable intellectually, but at that level alone.

To repeat, Dr. Richardson's formulation of structure, insofar as it goes, coincides with what has been previously described and repeatedly demonstrated. Dr. Richardson's concept of linkage of levels of functioning with levels of neurologic structure is interesting in view of the dynamic awareness that is being shown in psychosomatic medicine by both clinician and research workers in psychiatry and physiology. The field of psychosomatic medicine is too broad for any one individual adequately to study the complicated interrelationship, but I am hopeful that we will learn much from this new cooperative effort; truly expressive of the integration of psychiatry with medicine.

As for therapy, I fully appreciate the impossibility of presenting a lucid, practical discussion of this subject in the brief space of twenty minutes. Dr. Richardson speaks of therapy even more briefly, chiefly in terms of re-education by giving the patient insight into the structure and functioning of his personality. He says that there are many technical methods of accomplishing this objective synthesis, and mentions discussion, suggestion, free association, catharsis, and so on. He has, I believe, failed to mention the most important tool of psychotherapy, the ever-present relationship be-

tween physician and patient, teacher and pupil. The tool is probably the common factor in all schools of therapy regardless of technic, and is used constructively, knowingly or unknowingly, by all who achieve success in curative medicine. Is it not this phenomenon which explains the cures attributed to osteopathy, naturopathy and all the other pathies?

Probably Dr. Richardson considered this essential tool of therapy too obvious to mention, but I am anxious that he not neglect it, for it is a therapeutic necessity which every practitioner of medicine must keep in mind regardless of his field. The factor of the personality of the therapist himself and the patient's rational and irrational reaction to this personality is easily overlooked, for we, like the patient, may find this an unpleasant or painful awareness. This phenomenon was first described by the one already mentioned who ably formulated the structure of the personality. We may be original or otherwise and call transference and countertransference what we will, depending upon our pet rationalizations, but it is the cornerstone of every therapeutic relationship and consequently should be understood as to its character and usage.

If I have overstressed the omissions of this presentation, it is because I feel that those of us interested in psychiatric education need to clarify our formulation of the neuroses. We need to be scientifically honest insofar as we are able, and less satisfied with armchair philosophizing.

The common neuroses will probably continue to be treated in main part by general practitioners. Therefore, it is our responsibility to see that they are better equipped for this service. As long as the medical student is confused by vague abstractions and psychopathology which is too far removed from his own field of identification, he will continue to look upon psychiatry as something apart, and he will continue to look upon all neurotics as malingerers and treat them as such, leaving them to seek acceptance at a high price from the nonprofessional pseudotherapist.

Dr. Dexter M. Bullard, Rockville, Md.—I am very much in agreement with what Dr. MacDonald had to say in her discussion, because I do not believe that one can talk very intelligently about treating psychoneuroses. One does not treat a neurosis. Something does go on between a physician and a patient, and what goes on and under what circumstances the patient says various things and under what circumstances the physician replies and the attitude that the patient has toward the physician as he is and as the patient imagines the physician, and, conversely, the attitude the physician has toward the patient as the patient is and as the physician imagines he is, these are the things that are involved in the treatment of a person. One cannot treat a disease, but one can occasionally do something favorable with a patient.

I cannot see a great deal of use in jumping from structure to function and attempting to explain the social situation in terms of material, that is in terms of bodily structure. I think of a neurosis primarily as something which comes about by virtue of what other people do, and what the patient does in response; and to attempt to explain that by different levels of structure, to my way of thinking is no great help.

I cannot become very enthusiastic about lower and higher levels. I think that the ideals we have to leave to the ministerial group; that our function is primarily to see as accurately and as honestly as we can, to note what does take place, to help the patient to see what he is doing, and not to further either ideals or instinctive or reflex habits.

Dr. Spafford Ackerly, Louisville, Ky.—I remember working with Dr. Riggs for a short time, and I can say very definitely that Dr. Riggs, whether he knew it or not, very largely used a person-to-person relationship in his therapy. Dr. Richardson worked with Dr. Riggs for many years and he probably took that more for granted. He should not have taken it for granted, because it is something that does need clarification constantly. What is this person-to-person relationship at its best, at its worst, and all the way in between?

This paper is in line with advances in psychiatric thinking in the psychoneuroses and in psychosomatic medicine, which is not the field of psychiatrists any more, but of all medical men.

PSYCHOSOMA: THE MIND-BODY PROBLEM*

WITH SPECIAL REFERENCE TO THE GASTRO-INTESTINAL TRACT

By HENRY G. RUDNER, M.D.
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The achievements of modern science have so strengthened man's confidence in the power of his mind that he no longer shrinks before the occult nor holds the unknown in terror and awe. The dogmas and superstitions which obstructed his progress through many centuries have been exposed and overthrown, and in this age of enlightenment he boldly explores the realm of the supernatural, seeking to wrest from it secrets of more abundant life and health. He has already discovered an inseparable association between mental and bodily functions; now he is striving to map out the terra incognita of man's emotional way of thought, that those in whom the balance between these functions has been lost may be restored to an harmonious existence.

Today, the sum of knowledge in almost any field extends beyond the possibilities of one individual. In defense, therefore, we have divided this knowledge into numerous portions, and each of us, in an attempt at specialism, has devoted himself to the pursuit of a single portion. As a result of this so-called specialism, not only has the demand for mastery of all sciences become less urgent, but our restricted training has made it difficult for us to step out of our own particu-

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lar field. Yet, in proportion to our lack of general scientific information, especially insofar as it has a bearing upon medicine, are we, as doctors, handicapped in our efforts to evaluate and relieve human ailments.

The scientific study most closely related to that of the body is, of course, psychology. Particularly is it necessary that those who make a study of medicine familiarize themselves also with the functions and activities of the mind. In the past, medicine has been devoted almost wholly to a more thorough understanding of the organic aspects of disease. The fact that the mind has the power to influence bodily states has been disregarded, the majority of physicians preferring to treat specific pathologic conditions with a specific rationale. They have been distrustful of psychotherapy, relegating it to the province of the cults. During the past twenty-five years, however, an immensely greater attention has been directed toward the mind-body problem, as evidenced by the fact that the subject of psychosoma forms a massive part of present day literature.

Unfortunately, a number of medical men, in their enthusiasm over this new psychic approach to the problems of disease, now overlook somatic factors completely. The majority, by reason of their training, still regard disease as a strictly physical or organic disorder and treat it accordingly, allowing the patient's idea of his trouble to exert little or no influence on the diagnosis or management. Those who seek a psychic basis for every organic condition, on the other hand, are prone to take note of only an obvious mental or nervous element and, as a consequence, to tell such a patient, "You are nervous and merely imagine you are sick; go home and forget it." Often, the patient answers, "I know when I am sick," or "I have a pain and it is not imaginative."[14] These patients comprise a large part of every physician's practice and pay a goodly portion of his fees. But they exhaust the general practitioner. When all ordinary methods of examination fail to disclose a definite lesion, and routine laboratory findings are negative, they are labeled psychoneurotics and supposedly are shelved. Instead, they stumble into the hands of various specialists, are subjected to all kinds of refined diagnostic procedures, and often are again classed as psychoneurotics.[15] Still obtaining no relief, they finally turn to the various cultists and quacks.

Certainly, it is not to our credit that this should happen. Pain is one of man's most trying experiences. No matter in what part of the body, it takes precedence in his consciousness over all other interests, driving him to seek relief by every means available. Medical men should be able to provide this relief, if humanly possible, whether it be by medical or psychotherapeutic measures, or both.

The questions which confront us, therefore, are: (1) how has this condition come about; (2) what is really wrong with these patients, and (3) what are we going to do about it?

Originally, many of these patients have nothing more serious than a mild somatic manifestation of an anxiety. The real trouble starts with their first visit to the doctor's office. Observing in the course of examination an incautious remark of the physician, such as, "You have a heart neurosis," the patient begins to lose his psychic balance. Hearing "heart neurosis," he remembers the "heart" and not the "neurosis," and thereafter constantly feels his pulse, and in so doing increases his heart rate to an alarming figure. Eventually, he becomes a cardiac neurotic, developing, as a consequence, a long series of disorders.[4]

In like manner, patients become various kinds of hypochondriacs. For example, a physician's repeated measuring and recording of insignificant and transitory changes in blood pressure will so disturb a patient's psychic equilibrium as to cause a hypochondria hypertension. Or repeated urinalyses will fix his attention upon his urine report, bringing about a hypochondria analytica. Again, the prescription of an unnecessarily strict dietary regime may lead the patient to serious concern over the most minimal deviations in his diet and the belief that these deviations are responsible for his symptoms.[9]

The question, "Why do some individuals become ill under emotional strain while others do not?" is answered by the fact that people have different constitutional personalities. When a person collapses under emotional stress, we feel that something has inhibited his natural capacity to adjust himself quickly to extraordinary nervous stimuli. In such individuals, the threshold of resistance is lowered, and collapse occurs when stimulation is intensified until it crosses this threshold. A normal person accepts ordinary stimuli unconsciously, but when illness or pain is superimposed upon a lowered threshold, the sensory stimuli are quickly intensified, becoming pathologic sensations psychically elaborated.

To dispose of any indefiniteness or uncertainty as to the diagnosis of disease in these neu-

rotically predisposed patients, it is important that we study, not only the psychology of those whose illness is transient, but also the psychology of the chronically ill and incurable.[10] Undoubtedly, real organic disease is often masked by hysterical or neurotic manifestations. McKendree cites as an illustration of this fact the case of a woman who was irritable, selfish and insulting, who discharged physicians and nurses, and otherwise exhibited psychic phenomena. After a thorough search, she was found to be suffering with generalized carcinoma.[12] In such cases, we might more easily be content with noting the mental symptoms and classifying the patient's condition as a border line psychosis, but the possibility of overlooking some obscure disease should lead to an extensive physical examination of every neurotic patient, as well as a proper evaluation of all psychoneurotic manifestations.

Clinicians of today, therefore, should be cognizant of the fact that in predisposed individuals the earliest signs of visceral disease may be purely psychiatric in type, and, on the contrary, that psychic disturbances may produce adverse bodily changes. Viewed from this point, the diagnosis and treatment will often cease to be a problem.

Let us ask ourselves what we know at the present time about the physiologic effects of neuroses and how we can make use of this knowledge for the interpretation of neuroses. Most of you, I am sure, are familiar with Schilder's theory regarding vagotonia as first expounded by Eppinger and Hess; also, with the vegetative stigmatization theory of von Bergmann, and, more recently, the explanation of conditional reflexes by Pavlov. Likewise, Dunbar, Cannon, Barker and Fremont-Smith have contributed appreciably to our knowledge of psychic factors in the development of disease. Admittedly, most of us recognize symptoms produced by emotional strain, but we differ in our understanding of their significance. What really happens to the organs of the body as a result of continuous emotional stress? A change in organic function does take place. This is an accepted fact, both clinically and experimentally. For example, the patient with an ordinary case of angina, who tolerates usual exercise without inconvenience, will suffer an immediate attack following emotional irritation. Again, in peptic ulcer, emotional tension is often more harmful than dietary indiscretion. A settlement of the dispute as to the cause of epilepsy, migraine, Raynaud's disease, or Graves' disease may actually be found in psychophysiologic responses in emotional states.[8] Numerous articles by Crile, Stratton and Emerson, in which a correlation between emotional reactions and the incidence of disease is attempted, are bringing fruitful results in furthering our knowledge of the etiology of these diseases. Crile has aptly stated:

> "Just as worry or fright will produce white hair and a wrinkled face, which are physical changes, so may a distorted mind, through emotional disturbances, produce bodily disorder which may become a matter of importance itself."[5]

In this connection, Leopold Alkan has written a most illuminating and consistent book upon the psychoneurotic factor in organic disease.[1] This work has cleared up the vagueness and mysticism of psychic healing. Also, by explaining several facts which heretofore have been obscure, it has helped us in complying with the demand of pathologists and autopsiasts for morphologic evidence of disease.

Alkan emphasizes the existence of a psychic functional component which cannot be registered in terms of pathology, as well as the occurrence of anatomically demonstrable alterations which do not admit a psychic element. The manner in which the end organs react under the influence of vegetative stimuli depends upon their anatomic structure and the nature and extent of their various functions, such as contraction and secretion.

The tonus of only a few striped muscle complexes, such as the diaphragm and external sphincters of the intestine and bladder, is affected by adverse stimuli of the vegetative system. Of more serious import are the alterations in function of the smooth muscles of the blood vessels, which are also psychically conditioned. Contractures of these structures may lead to a number of anatomic disorders. For example, a lasting contraction or spasm of a hollow organ produces compression and consequent anemia of the region involved, so that malnutrition and pathologic changes ensue. Peptic ulcer and latent ulcerative colitis are illustrations of this process. In like manner, repeated spasm of tubular organs may lead to hypertrophy and permanent dilatation, as in essential hypertension, hypertrophy of the left ventricle, and dilatation of the esophagus, or to partial paralysis and stasis followed by pathologic changes and an alteration of concentration or fermentation of the contents of the organ. The gallbladder containing cholesterin stones proves the latter transitions.

When secretory functions, especially of the

endocrine glands, are affected by excessive vegetative stimuli, such pathologic entities as diabetes or Graves' disease may be observed.

I wish to acknowledge here my indebtedness to H. Flanders Dunbar's comprehensive review of the literature dealing with the interdependence of mind and body in pathologic changes, from which I have taken the liberty of quoting freely.[6]

The gastro-intestinal tract was one of the first functional systems upon which the influence of vegetative stimuli was recognized. Both gastric motility and gastric secretion are affected by emotion. Spasm of the small intestine, especially segmental spasm, and the mechanism of ileus following minor operative procedures are familiar to all of us. Indeed, the effects of excessive stimuli on both the physiology and function of the digestive organs are so frequently encountered that it seems worth while to devote particular attention to this phase of the psychosomatic problem.

Barker defines spasm of the colon as an enteropathy affecting nervous patients. Jordan regards it as a condition in which the musculoneural apparatus has lost its coordination and correlated function. Bastedo[2] states:

"There is much difference of opinion about its character, and no agreement about its etiology."

Many writers, however, among them Bargen, Jordan, Bokus and Brown, agree that it is purely neurogenic, and that the large quantities of mucus produced are hypersecretions rather than inflammatory exudate. To quote McLoone:

"It is rather the routine of the individual superimposed upon a none too stable nervous mechanism that produces the 'irritable bowel,' and the condition, once established, works in a vicious circle."[13]

Those of us who have followed the pioneers in gastroenterology and physiology and have scrutinized the correlations of the clinician, physiologist and biochemist feel that we have weathered the storms and revolutions in medicine and are now face to face with physiologic truthfulness. To such men as Alvarez, Howell, Carlson, Hurst and Cannon we are deeply indebted for our knowledge of the physiology of the gastro-intestinal tract. This tract is certainly a most intricate piece of machinery. Although it would seem illogical to compare this machinery with that of the heart, nevertheless, it has an almost identical nervous mechanism.

In the gastro-intestinal tract the nervous mechanism is initiated by the ingestion of food. When food enters the stomach the pylorus closes, and a signal is sent to the ileocecal valve, which opens to allow the fluid medium held in the ileum to pass into the cecum. A contraction of the rectosigmoid ensues. This is the gastrocolic reflex, and is a normal response which occurs after each meal. When the fluid enters the colon, it causes the mass in the colon to move by direct physical effect and not by peristalsis.

The clinical picture of the irritable colon may be found in persons of every age and type. It is seen in children 8 or 10 years of age, and in adults of 70. It prevails among both sexes, although women are more susceptible than men. It is equally common to people of all races. The highly sensitive, hyperesthetic types, such as musicians, artists, students and professional men and women, are likely subjects, as well as those with a neurologic background. The majority come from allergic families and are themselves definitely allergic, presenting manifestations of hay fever, migraine and urticaria. Many suffer with chronic sinus infection. Others have endocrine disturbances, such as dysfunction or enlargement of the thyroid gland. Approximately 20 per cent of the women have dysmenorrhea. Myalgia, neuralgia and neuritis are commonly associated complaints. All these patients, in fact, may be said to have an inferiority of the nerve mechanism of the gastro-intestinal tract with a potential low threshold to sensory stimuli.

In order to evaluate more clearly the symptoms of the irritable colon, it may be well to classify them according to three types: (1) the mild, (2) the moderately severe, and (3) the severe.

The Mild Type.—The patient with only a mild degree of colon irritation complains chiefly of constipation. His stools are few in number, small in caliber, sometimes flat in contour and at other times resembling sheep dung. Also, he has periodic headaches, takes barbitals at night in order to sleep, and gains and loses weight. He is addicted to enemas and purgatives, is a frequent visitor to spas and dietetic institutions, uses a variety of dietetic foods, and in fact makes a study of foods. Finally, when these measures fail, he consults a physician.

When first seen by the physician, this patient has already become introspective and self-analytic, and his entire interest centers on his abdomen. He complains of a burning sensation in the abdomen, gaseous distention and pain. The pain may be either acute and paroxysmal in type or merely a dull ache. It may be general over the abdomen or localized to one region.

If in the epigastrium, it may be referred to the left shoulder, as in gallbladder disease, or may occur after meals, indicating peptic ulcer. The pain is inconstant, however, occurring after some meals and not after others, after some foods but not after others, and on some days though not on others. It is not periodic, and never does it awaken the sufferer at night.

A large number of these patients complain of pain in the ileocecal region. I have found this true in over 40 per cent of my cases. Jordan and Kiefer report that 25 per cent of their patients with irritable colon had their appendices removed,[11] and Eggleston states that 22 per cent of his patients similarly affected had appendectomy.[7] Although undoubtedly there are cases wherein a differential diagnosis between appendicitis and disorders of the colon is difficult, and even after observation a laparotomy may be justified, these figures are proof of an all too common haste in removing the appendix. As Bettman says:

"Where appendectomy is performed on the patient with the diagnosis based on nothing more than indigestion and a poke in the right iliac region, it is no wonder that the series is so high."[3]

The Moderately Severe Type.—Here, the patient has all the symptoms of an irritable colon in an aggravated form, including flatulence and gaseous distress, with absorption of absorbable gases.

At this stage the patient begins to lose weight, is nauseated, vomits an occasional meal, and develops a disturbing insomnia, apprehensiveness, mental depression, and psychoneurosis. A symptom complex of neurocirculatory asthenia, with sweating palms, tachycardia, tremor, hot and cold flashes, vertigo, spots before the eyes and tinnitus aurium is observed. Precordial pain, similar to that of angina, may be present. Frequently, a bladder disturbance, manifested by ureterospasm and retention of urine, is associated. Usually, the temperature is irregular. Attacks of abdominal distress occur with increasing frequency and the patient refuses to eat, or develops Osler's symptom complex, anorexia nervosa. Eczema or other skin lesions may appear, and the patient's skin takes on a sallow or dusky hue. The picture in this type can resemble a number of diseases, such as pellagra, pernicious anemia, carcinoma, sprue, or miliary tuberculosis.

The Severe Type.—When the colon has become irritable to a severe degree the patient requires hospitalization. As a rule, he presents evidence of acidosis or alkalosis, marked dehydration, and avitaminosis. The skin is dry and exudative, the eyes are sunken, the tongue is red. He is unable to take nourishment, necessitating feeding by tube and venoclysis. He may have hyperemesis and diarrhea in the extreme, and convulsions are not at all uncommon. Pyrexia is the rule and not the exception. It is here that we see the so-called gut leukocytosis, with a leukocyte count of 10,000 to 20,000. Urinary suppression ensues, and only the most drastic procedures can save the patient's life, such as blood transfusions, and the intravenous use of glucose or, if alkalosis is present, hypertonic solutions of sodium chloride.

Not infrequently, the patient presenting gastro-intestinal symptoms falls into the hands of the surgeon between the second and third stage, or during the third stage. This is truly hazardous unless the surgeon is thoroughly familiar with the condition and can make a correct diagnosis. The patient has an even chance with surgery during the first stage, as his resistance then is not seriously impaired and he can usually withstand fairly well the insult to his abdomen. When the third stage has been reached, however, the surgical risk is indeed grave.

Since gastro-intestinal disorders are frequently the first signs of emotional imbalance, it is to the internist or gastroenterologist that these patients come first for relief. In the absence of physical and laboratory evidence of disease, a definite relationship can usually be established between the patient's complaint and his nervous mechanism, through information elicited at repeated interviews regarding his personality and environmental background. Once such a relationship is established, the somatic manifestation is, as a rule, easily brought under control; when the patient acquires a thorough understanding of the source of his symptoms and develops a resistance to emotional disturbances, he promptly improves. In such cases, obviously, the internist and gastroenterologist must assume a double responsibility: the patient must be treated not only medically, but psychiatrically as well; not merely as an irritable gastro-intestinal tract, but as a total individual.

After all, this method of approach is not new. Rather, it is a revival of one of the oldest axioms of medical practice. Socrates voiced the idea over 2,000 years ago:

"Just as you would not treat eyes without head, nor head without body, so you would not treat body without soul."

We of today can display no better wisdom in the treatment of gastro-intestinal disorders, as well as of many other afflictions which assail the human species, than to follow his counsel.

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ACNE VULGARIS A PUSTULAR LIPOIDOSIS*

SUCCESSFUL TREATMENT BASED ON CONTROL OF LIPOID METABOLISM BY LOW FAT DIET AND THYROID EXTRACT

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The Problem.—The various treatments proposed for acne seem not purposively designed. Little agreement is found among many authors, excepting (1) that roentgen radiation helps by disabling sebaceous glands, (2) that sulfurous astringents have a beneficial effect locally, (3) that diet—some sort of diet, but none of them has been consistently planned—is a good idea, and (4) that removal of a comedo may prevent the development of an acne pustule at that place. *Corynebacterium acnes* has been found in acne, and it has been found also in normal skin. Acne pustules are not inoculable as boils are; acne does not behave as an infectious disease. Vaccinal "etiologic" treatment[1] with *C. acnes* was once popular, but nowadays vaccinal therapy has fallen into disuse.[2] Acne has something to do with hormones,[3 12b] with glands of internal secretion, with pubescence, and perhaps with pigmentary function. Heredity[4] predisposes. The disease may be familial.[5] Carbohydrate metabolism[6] has little if anything to do with it. Bacteria probably have nothing to do with it. *Demodex folliculorum* commonly leads a saprophytic existence in sebaceous glands.

Facts which seem to me significant in trying to formulate an etiologic understanding of acne are these:

(1) Seborrhea precedes the development of acne, and conspicuously accompanies it.

(2) Comedones and milia accompany acne, but they may exist without the development of inflammatory lesions.

(3) Acne has something to do with diet.

(4) The patient with acne is usually one who can scarcely get enough sleep, but who, after 9 hours abed, is hard to awaken, and also feels tired later in the day.

(5) The patient with acne is often constipated, is generally depressed emotionally, and, if a woman, is likely to have menstrual difficulties and peculiarities.

(6) An acne pustule clumsily squeezed becomes a bigger, cystic sore.

(7) An acne lesion of the deeply seated, cystic, inflammatory sort is made worse by every kind of local treatment, including incision and roentgen therapy, but it responds best to being let alone for several weeks.

(8) Vaseline (and other oily substances[7]) promote acneform lesions when rubbed on the skin. One distinguishes staphylococcic folliculitis (boils), which are encouraged and spread by greasy applications, from acneform lesions due to paraffin and other oils.

(9) Iodides always make acne worse.[8] In some non-acneic persons, iodides provoke the appearance of acne. Other persons, on the other hand, are "normal," and tolerate iodides in considerable dosage without developing acneform eruptions. Iodides given in toxic hyperthyroidism do not provoke acne.

(10) The patient with acne is usually a person who is undergoing the changes of puberty.

(11) Acne is worse in the summer time. It undergoes irregular and seemingly unpredictable exacerbations,[9] new lesions appearing in crops. It is usually made worse by intercurrent illness and by menstruation.

(12) Comedones and acne vulgaris appeared

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in many children of two children's homes while I was observing the children closely. Both epidemics of acne followed the routine administration of cod liver oil to all of the children.

(13) Acne often appears soon after young women try to gain weight by drinking cream in large quantities. Ice cream and chocolate provoke exacerbations of acne, as every patient knows, or learns.

(14) Acne appears with notable rarity in avitaminosis A.[10]

(15) In a patient's skin which shows plain evidence of chronic roentgen damage, seborrhea continues and inflammatory lesions flare from time to time.

(16) A well defined clinical group of acne cases comprises about 10 per cent of all cases. In these the lesions are rosy, rosacea-like, or better xanthoma-like, and occur with oily seborrhea and few comedones in persons who are usually not "tired" as the average patient is, and whose hair coloring has a reddish tint.

Etiology.—The statement of my theory is reduced to its outline. Seborrhea is excessive oiliness of the epidermis and its glands. Comedones and milia are collections of oily matter. An acne lesion, shallow or deep, is a foreign body reaction to oily epidermal material,[11] which may be liquid or solid at body temperature. The whole process represents failure of the bodily chemical economy to manage oils successfully. It has nothing to do with parasitism. Seborrhea, comedo and acne vulgaris are analogous to diabetes mellitus. In diabetes, excess of carbohydrate collects beyond the ability to metabolize it; diabetes is controllable by reducing carbohydrate intake, enhancing carbohydrate metabolism, if necessary, by giving insulin. In acne, excess of lipoid collects beyond the ability to metabolize it, and provokes pustular foreign body reactions; acne is controllable by reducing lipoid intake, enhancing lipoid metabolism, if necessary, by giving thyroid extract.[18 19]

Acne vulgaris may be defined, in my opinion, as the disease which consists in inflammatory reaction to sebum, which is excessive through the subject's improper metabolism of lipoid. It is a pustular lipoidosis. In contrast with the mesodermal lipoidoses (xanthomas), acne vulgaris is a primarily epidermal lipoidosis, with secondary inflammation. Acne vulgaris depends (1) on the excessive dietary intake of lipoid (and/or lipochrome substances); (2) on hormonal influences, through the function of the thyroid gland in the control of lipemia,[18 19] thyroid function being itself dependent on other endocrine apparatus and their balance and integrity; (3) on vitamin intake, through (a) the need for vitamins in maintaining epithelial integrity, and through (b) the antagonism[28] between carotinoids and vitamin A on the one hand and thyroxin on the other; and (4) on iodine, (a) in the excess of which hyperlipemia occurs in hypothyroid individuals, but not in normal ones, and (b) in the absence of which the thyroid gland cannot long function normally.

Sensitivity to iodide[8] on the part of the patient with acne is due to his hypothyroidism; give the acne patient iodine and thyroid extract in adequate amounts and he does not suffer eruption. I allow my patients iodized salt, and it is tolerated.

There are many reports of attempts to control acne vulgaris by various hormonal substances. Without reviewing them here, I believe it fair to say that results have been irregular and unsatisfactory. Hormonal therapy[12b] in the past has erred because it has missed the point of need for balance between diet, metabolism, autogenous hormonal activity and medicinally supplemented hormonal substances. I repeat the analogy between diabetes mellitus and acne. What good would insulin do if the diet were not simultaneously and accurately considered? Some cases of acne can be controlled with a strict low-fat diet. Some cases have been helped by hormones alone. Almost all cases can be controlled with low lipoid diet supplemented by thyroid extract given to tolerance.

Some cases I[13] was not able to treat with success in this way. These unresponsive cases are all of one type (see paragraph 16 above), being cases in which comedones are tiny or absent, in which the lesions are rosacea-like and similar to xanthomas, in which the lipoid is liquid at body temperature, in which the individual is red-haired and has a skin of exceptionally fine texture (that is, small fat glands and thin epidermis). These cases comprise a group that seems to bridge the distinction between acne vulgaris and xanthoma. They are patients who, in general, are not benefited by roentgen therapy, either. These cases, as I determined by clinical experiment since 1939, are responsive to a low-carotinoid diet.

Seborrhea is the cutaneous manifestation of failure to metabolize oil. It is a feature of acne vulgaris throughout, for it is a manifestation of the basic pathologic physiology. Comedones

and milia represent the retention of sebaceous matter within distended but atrophic skin glands,[11c] which may or may not provoke foreign body reaction. Bloch[3] defined the two stages of acne, and wrote:

"Only the [comedo] phase of acne can be brought into direct relation with endocrine processes in sex glands. The second phase of acne is characterized by an inflammatory and suppurative process on a basis of infection. It may follow the first phase, but this is by no means inevitable."

If the inflammatory phase resulted from infection with *Corynebacterium acnes*, which is almost invariably present, the second phase ought to be practically inevitable. Since the second phase is not inevitable, this is a reason for believing the bacterium a saprophyte. Bloch assumed that inflammation implies infection; thus he overlooked the fact that the inflammatory phase of acne is foreign body reaction to lipoid,[14] an inflammatory reaction with phagocytes and giant cells which has induced pathologists to confuse acne with tuberculosis,[11b] a reaction which is the inevitable result of introducing fatty material into mesoderm. It can result from bruising a comedo, from injuring a sebaceous cyst, and from injecting into the skin chemical fractions of *Mycobacterium tuberculosis* or chemical derivatives of olive oil or of cod liver oil. I hypothesize that decadence of glandular epithelium (which should separate sebum from mesoderm) occurs as a result of the presence of the comedo, which, being lipoidal, dissolves lipoids, including vitamin A, preferentially from the epithelial cells adjacent to it. Cod liver oil, rich in unsaturated fatty acids, causes the development of comedones in adolescent children who are given the drug in excessive quantities, but inflammatory lesions are few in these children. In them, I believe, the integrity of the epithelial barrier is maintained. But when the comedo results from the hyperlipemia of hypothyroidism, with its attendant malmetabolism of vitamin A, then the epithelial barrier is likely to become defective, and inflammation is likely to complicate the presence of the comedo.

A carefully designed low-fat diet, conscientiously followed, is promptly effective in diminishing seborrhea. While untoward symptoms of lack of unsaturated fatty acids occur in rats on a strict low-fat diet, a human being on the same diet[15] suffered no damage, but actually felt the better for it. I have seen no ill effects from the low-fat diets that have been followed under my supervision by more than 400 patients with acne, but they have been relieved of fatigability, constipation, and mental depression to a noteworthy degree, while at the same time their acne gave ground.

The constitutional symptoms of the patient with ordinary acne are due to his relative hypothyroidism;[16] by "relative" I mean the inadequacy of his autochthonous hormone to cope with the quantity of lipoid he has been ingesting. Constipation is no cause of acne; the acne patient is constipated for the same reason he has acne. Menstrual difficulties in acne are usually on the same basis, and so are anemia and "that tired feeling." These are all accompaniments of hypothyroidism.

Squeezing pimples in a clumsy fashion makes them worse because trauma crushes droplets of lipoid throughout the inflamed region. Each particle of lipoid, liquid or solid, superficially or deeply situated, then does its share of provoking irritation. Deeply seated inflammatory lesions are lipoid-filled granulomas. Try rubbing between your fingers their greasy, sanguinolent contents. To incise such lesions does no good, for only a fraction of the matter drains out. Roentgen therapy does no good, for it merely liquifies the contents by necrotizing phagocytes, and it frees oil and enables it to irritate the more (the well known "explosive" effect[17]). If one simply leaves such lesions alone, they eventually resorb, for the oil eventually is either phagocytized and carried away, or it becomes encapsulated within epithelium so as to form a sebaceous cyst.

Paraffin acne is the result of foreign body reaction to matter forced into follicles and glands. Some substances, such as chlornaphthalene,[7] are much more irritating than others. Hass[11a] demonstrated in his studies of the results of injecting esters of fatty acids of olive oil and cod liver oil that nonsaponifiable fractions are severely irritating and that intensity of inflammatory response increases with increase in average unsaturation of the lipoid fraction.

The influence of thyroid extract on lipemia has been studied especially by Hurxthal,[18a, b] who showed that blood lipoid determinations give a sounder estimate of thyroid function than the basal metabolic rate. Turner and Steiner[19a] followed blood lipoid levels in a number of individuals and demonstrated that thyroid extract always diminishes cholesteremia markedly, while iodide increases it only a little if at all, affecting only some individuals. Others[10b, c] have observed the marked influence of thyroxin on lipemia.

Investigations of cholesteremia by Strickler and Adams,[20] in which were reported 217 determinations of cholesterol in 103 patients with acne vulgaris, were interpreted to indicate that there is no correlation between lipemia and the disease. This report is a misapplication of statistics, for the averages are arithmetically sound but medically meaningless. Three or four determinations at intervals of several days or weeks in 100 patients give little hint of what information would result from 100 determinations at intervals of a few hours in three or four patients. The average would be unimportant, just as averages of the several figures comprising a glucose tolerance curve would be; it is the peak that counts, for it is at the peak, I believe, that oil is "spilled" into the sebaceous receptacles, after which, within 24 to 48 hours, acneic inflammatory reaction becomes apparent. But cholesteremia may be normal and yet the fat metabolism deranged.[21] Furthermore, investigations of acne patients' free fatty acid, carotinemia and total blood lipoids have not as yet been made. Fatty acid levels[10c] are probably more important than cholesteremia. Lipoid tolerance curves should be drawn for acne patients, analogous to glucose tolerance curves for diabetics. When all of the lipoid fractions are so investigated (not merely the cholesterol fractions) then, I predict, lipoid metabolism will be demonstrated to be deranged. I often see that a patient's acne erupts the day after he eats ice cream. Plainly, oil that has been swallowed provokes inflammation typical of phagocytosis of lipoid in the skin of the face; it gets there somehow. Take the oil from the food and inflammatory incidents become fewer, but cystic lipoid deposits become inflamed from time to time; then take the oil from the deposits by means of a bistoury and gentle technic, and few inflammatory incidents thereafter occur. Give thyroid extract to help burn oil if altering the diet is insufficient to bring intake within tolerated limits; then the patient generally gets well and stays well. That is, 90 per cent of the patients do. In the remaining 10 per cent of the xanthomatoid class a strictly low-carotinoid diet in conjunction with a diet low in animal fat is helpful.

The adolescent is especially vulnerable to acne because often he is inadequately[16a] supplied with thyroxin. Perhaps the development of the thyroid gland may not keep pace with demands made upon it. The young individual furthermore is one who customarily has pushed into him, by well-intentioned parents who believe the advertising, quantities of milk and cream far beyond his ability to metabolize their oily constituents. Of milk, 70 per cent of the caloric value is lipid.

Acne is worse in the summer time because of (1) gross increase in oil intake on account of the popularity of ice cream; (2) increase in seborrhea because of the direct effect of warmth and sweating, and (3) liquifaction of comedo oil, making it mechanically easier for sebaceous lipoid to reach mesodermal tissues capable of reacting to it with inflammation.

Rarity of acne in avitaminosis A[10a] occurs because (1) avitaminosis A occurs only in persons on extremely low-fat diets and (2) keratinization of epithelium in avitaminosis A prevents the conjunction of sebum and mesoderm, which is the *sine qua non* of an inflammatory lesion, while hyperkeratosis[10b] damages the epithelium of the sebaceous gland itself so as to diminish seborrhea.

Sulfur lotions are useful astringents, giving the same sort of local symptomatic relief that x-ray does. There is some connection between sulfur and the metabolism of vitamin B complex,[22] which may or may not be pertinent here. But sulfur lotions are not necessary in treating acne; my cases do as well without them as with them.

Roentgen therapy, short of necrotization, cannot remove a depot of lipoid from the skin, particularly if the lipoid is within an intracutaneous or subcutaneous, epithelium-lined cyst. Roentgen energy can destroy sebaceous glands, but this does not permanently prevent epidermal oiliness if lipoid metabolism is not controlled. Faces showing x-ray atrophy often remain seborrheic, and collections of lipoid set insularly within atrophic skin can provoke foreign body inflammation from time to time. Nor can roentgen therapy permanently incapacitate mesodermal tissues from reacting to lipoid, for phagocytes and histiocytes reappear eventually. Roentgen therapy should be used in acne only, in my opinion, after lipoid metabolism has been set right, some weeks after the patient has started dietary and other therapy. Then the use of roentgen therapy usually is unnecessary. Roentgen therapy will not cure acne when no account is taken of the basic pathology of the disease, for it treats the symptom, seborrhea, not the cause, which is defective lipoid metabolism. After lipoid metabolism is attacked, insofar as I can set it right by diet and thyroxin, then, after about two months of treatment, my patient may receive some four biweekly unfiltered doses of 150 r each.

Treatment.—Meticulous instruction is necessary. The patient must avoid traumatizing the face. In washing, any nonmedicated soap will do; it is to be used gently, with tepid water (not hot, which would melt the oil in the lesions). Sore lesions are avoided rather than massaged. No unguent of any kind may touch the face. Vaseline on the hair gets on the face. Powder (light and fluffy), rouge (not cream rouge) and lipstick are allowed.

Seborrheic dermatitis of the face, "dry skin," is the extension of dandruff from the scalp. One should treat the scalp with a suitable antiseptic, such as a resorcinol monoacetate lotion, so that scaliness will not interfere with secretion of sebum.

No local medication is prescribed for the face, not even sulfur lotion.

Local treatment of comedones and pustules must be performed by the physician, the patient being prohibited from picking at his lesions. The patient is ordered not to touch the face at all; for Stokes and Garner[23] showed the importance of postural habits, such as leaning the chin in the hand, in promoting localized acne eruptions.

Local treatment, as performed by the physician, is designed to get rid of lipoid depots before they provoke inflammation or with as little trauma as possible after they have done so. Sebum surrounded by epithelium will remain unabsorbed indefinitely. Sluggish, inflammatory phagocytosis of sebum gets rid of it only slowly, constituting the acneic pustulation which one wishes to avoid. By skillful removal of comedones, scarring is minimized. The patient is requested to make weekly visits for such local treatment, which is performed patiently and carefully. Removal of comedones is important, but it must be done without crushing particles of lipoid into surrounding tissues. The uninflamed comedo is often bulbous, so that a tiny "episiotomy" (from the mouth of its follicle outward along the line on the skin of the geometric projection of its slanting axis) enables one to pop it out without applying undue pressure. A comedo surrounded by inflammation must be treated exceedingly gently; if, after slitting the follicle, it does not come forth readily, it is better let alone than bruised. Solid lipoidal collections are far more easily removed than liquid ones, and a cold pack is more suitable as a preliminary in cases of low-melting-point seborrhea than the hot pack which makes it easier to remove solid comedones. The pustule containing a semidigested comedo must be slit open just widely and deeply enough to let out the contents.

The diet contains ample amounts of those five classes of ingestive substances which collectively are both necessary and sufficient: water, minerals, proteins, calories, and vitamins. Calories are obtained from carbohydrates, not from fats. There is no limitation of caloric intake. Dietary instruction is given with great care, pointing out to the patient that in general foods of animal origin are oily while foods of plant origin are not. Allowed are cereals (hot or cold, including wheat, including bread), fruits, vegetables (every vegetable substance excepting nuts, chocolate, and vegetable cooking oils), sugar (and sugary things like jam, honey, syrup, and sugar candy), salt, pepper, spices, flavorings, and finally lean meats in comparatively small quantities. While my diet has been criticized as being prejudicial to well being, the truth is that this criticism is based on hypothesis, not on experiment. My patients are simply on a high carbohydrate diet. Enough vitamin A, the requirement for which is not great,[15b] and the dearth of which is not startlingly detrimental,[10] (unless the lack is severe and prolonged[15b]) "leaks" into any diet that an ambulatory patient selects for himself. I assert, on the basis of four years' experience with many patients, that a low fat diet is not harmful.

The diet[24] is as follows:

LOW-FAT DIET

Do not eat these fatty foods

Milk
Cream
Ice Cream
Butter
Butter substitutes
Cheese (cottage cheese O. K.)
Lard, crisco, cooking oils
Shortened foods (pie crust, biscuits, crackers, etc.)
Fried foods (potato chips)
Pork
Ham
Bacon
Sausage
Chocolate and cocoa
Nuts and peanut butter
Cod liver oil
Vitamin A concentrates (B is O.K.)
Cream soup
Gravy
Catfish, Spanish mackerel, smoked herring, shad, canned fish, salmon, tuna, sardines, mutton, brains, yolk of egg, oatmeal

Eat these nonfatty foods freely

Cereals; bread — stuffs made with little shortening
Lean meats (including beef, chicken, sweet breads, kidney, bass, cod, flounder, haddock, perch, pike, snapper, brook trout, shrimp, frogs' legs, clams, oyster, lobster), gelatin and white of egg
All vegetables (excepting olive, nuts, avocado and those noted in low carotinoid diet for certain cases)
All fruits (excepting as noted in low carotinoid diet for certain cases)
Sugar, preserves, jam, jelly, honey, molasses, syrup and candy made of sugar (but not of butter, nuts, chocolate), salt, pepper, spices

LOW CAROTINOID DIET (for cases mainly of the rosacea-like, xanthoma-like type)

Avoid	Allowed
Carrot	Tomato juice; catsup
Orange (two a day allowed)*	Beets
Pumpkin	Colorless and lightly colored vegetables and fruits.
Sweet potato	Beans, peas, corn, grapefruit, pears, apple, banana
Spinach	

Instructions

Iodized salt may be used in cooking. Iodine is an essential food element, in the absence of which trouble with the thyroid gland is inevitable. Iodized salt is not necessary in some regions.

One must follow the diet strictly and continually.

The diet is low in calcium. In pregnancy, calcium must be provided.

The diet is low in vitamin A, on purpose, and harmlessly for a period of 6 months.

I have never found a patient unwilling or unable to follow the dietary plan pretty successfully, although people who eat only at restaurants have difficulty avoiding grease.

Thyroid extract is given to all cases and is given to tolerance[25] on clinical criteria without regard to determinations of basal metabolic rate or blood chemistry. Almost all acne patients tolerate at least a small dosage. Coffee, tea, and coca-cola (0.6 gr. caffein per bottle) mask symptoms of hypothyroidism. In chronically fatigued, thin, worried patients, who fight their "tired feeling" and suffer insomnia in consequence, the medication improves sleep, rids them of their nervousness, and helps them to gain appetite and weight. I habitually prescribe one 2-grain tablet of desiccated whole gland substance† each day with the evening meal, and may increase this dose to 2 tablets a day, one with breakfast and one with dinner. Sometimes more is needed. The dose must be estimated by clinical trial, giving just less than the amount determined by experiment to produce symptoms of excess. The dose must be adequate. After a few weeks, the need may have been made up, so that a smaller dose will maintain; sometimes my prescription is to take the tablets for two weeks, then diminish the dose by half. I have seen no ill effect of overdosage which did not pass off in a few hours, without a trace beyond the memory of it. While all acne patients tolerate at least a small dose, and many patients require surprisingly large amounts before symptoms of intolerance appear, yet any dose given a normal person causes a sense of tension and exhilaration, likely to be followed by a violent headache like that which is the aftermath of anger. In a normal person, any additional thyroxin is too much. Headache, tremor, palpitation, insomnia and nervous exhilaration are indications for immediately diminishing the dose. Emotional instability and continued weight loss after 3 weeks are other indications for reducing the dose. I give maximum tolerated dose; if a dose is tolerated with not one symptom of overdosage, it must be harmless. The patient is questioned at each weekly visit and the dose is adjusted. The patient is told about symptoms of overdosage and is ordered to stop taking thyroid medication at once if any symptom of overdosage appears.

One should discourage free sweating, for to sweat is to secrete also an increased quantity of sebum, and to exert violently is to devote thyroxin to this purpose when it might be utilized otherwise to greater advantage. I excuse my patients from "gym."

Adequate sleep is desirable, for chronic fatigue is certainly harmful. Often the fatigue which is chronic and scarcely relieved by sleep responds excellently to small doses of thyroid extract.

Erotic preoccupation is to be avoided, as Cleveland[26] recommended. This is a difficult prescription to put across, but it is probably sound advice. I sometimes see acne disappear after marriage, and see it reappear, particularly accompanied by neurotic excoriation, after divorce.

Results of Treatment.—Objective improvement, diminution of seborrhea, is visible in a week. Improvement is obvious in a month, is excellent in six months. Improvement in symptoms such as mental depression and fatigability occurs in a week, and is striking in most of the patients. A weight loss of 3 to 10 pounds may be expected in the first month, especially in large scale milk-drinkers. Weight loss is counteracted by eating more of the allowed foods. Weight loss ceases after four weeks and weight is eventually regained. This loss is no cause for worry. Appetite is greatly increased; for the low-fat diet is bulky and does not "stick to the ribs." Constipation disappears.

Some three to six months are required for cure. The diet is tentatively broken after a few months when the skin has become normal, enriching it particularly in vitamin A. The patient thereafter determines by experiment what his lipoid tolerance is.

Comment.—After some study of the nature of proof and of the transient and temporal charac-

*Dr. Stuart Way told me (November, 1939) that, in his practice in San Francisco, he had restricted the ingestion of oranges by his patients with acne, on empiric grounds.

†Burroughs-Wellcome & Co.

ter of many of the "proofs" which have been submitted for various scientific concepts, I have come to believe that proof simply is that which is convincing; in the interpretations of biology, absolute proof is difficult to arrive at. I have tested my theory in three ways: (1) by its logical coherence; (2) by its consonance with other investigations concerned with lipoid metabolism, which fit into my theory like parts of a jigsaw puzzle, and (3) by the effectiveness of treatment based on it.* Laboratory and clinical tests can easily be devised. One clinical test of common experience is performed by slender young women who undertake to gain weight by drinking large quantities of milk and cream; many of them promptly develop acne. Thyroid extract has been used in the treatment of acne by many physicians,[27a b] but my recognition of the necessity for balance of lipoid metabolism in acne patients by using diet and thyroid extract for the reasons given is original. I have recognized only recently the harmful effect of lipochrome pigments (including carotinoids) in the 10 per cent group of rosacea-like cases.

In testing this method of treatment, I trust that, in cases of apparent failure, one will seek for error in the administration of it, or in the way the patient follows it, before condemning the approach as a false one. Some cases I failed to relieve when I began applying the method in 1937 were relieved later when I learned how to keep the patient on a truly low-fat diet, and again when I excluded carotinoids in some cases. Actual cooperation by the patient is requisite.

SUMMARY

Acne vulgaris is a metabolic disease. It depends on imbalance between the dietary intake of lipoids (and/or lipochrome substances) and the patient's capacity for metabolizing them.

Acne vulgaris is, histologically, foreign body reaction to lipoid; this inflammation is not bacterial inflammation.

Milk and milk products, being particularly rich in fat, are the commonest harmful ingredients of the diets of patients with acne; their baneful effect has nothing to do with allergy.

The details of a practical and successful method of treating acne vulgaris are described and explained. This treatment depends on three factors: low-fat diet, suitable dosage of thyroid substance, and mechanical evacuation of lipoid deposits from the skin.

Danger to nutrition inherent in such a regimen is not a practical deterrent if the patient is kept under the control of an alert physician.

*As Bacon has said, sciences are judged by their fruits, and in medical practice the outcome of treatment is no less a test of the doctor's use of his conceptual scheme, and in the end of that scheme itself, than is the outcome of a laboratory experiment a test of the theory of the experiment (Henderson: Science, **94**:1, July 4, 1941).

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DISCUSSION (*Abstract*)

Dr. M. T. Van Studdiford, New Orleans, La.—Care should be taken to limit discussion to frank cases of acne vulgaris, avoiding acnitis and colliquative tuberculous lesions, which usually react to the bovine tuberculin test. This should be done to protect the general practitioner from rushing headlong into the two methods of therapy suggested without proper diagnosis of the condition he wishes to treat.

The different loads that cause acne when they are dumped on certain individuals are carried along so many roads that finally converge that in approaching the treatment of a case of acne I stand at the crossroads myself, looking down each road, trying to determine by which one the condition has arrived.

Dr. Sutton has brought out the fact that one side road which must be investigated is what goes on in the sebaceous gland to produce acne. Whether we believe that a sebaceous gland secretes or breaks down to form the material we find coming from its orifice, we all believe that the thyroid, and other glands of internal secretion, have an effect upon the sebaceous glands. How and why these glands are upset is the problem to be solved. Basal metabolism determinations are usually lower in the winter, and, as Dr. Sutton has said, acne gets better in the winter. Heat and cold, sun and winter darkness, fat intake in summer when less is needed, all may cause the increased or decreased activity of sebaceous glands. One can see offhand that increased thyroid activity will compensate for some of the extra fat intake in summer.

Another consideration would be the nervous stability of the patient and his physical inheritance. An individual inherits certain proclivities of the thyroid gland just as he inherits certain tendencies of the sebaceous glands, and much stress can be put upon their reactions under different stimuli. We cannot change the individual's physical inheritance, so we are forced to treat what that inheritance will present.

A fat diet or a non-fat diet will of course be tried by many of us.

I would probably be considered radical or out of step if I should say that I use cold cream or corresponding cleansing ointments upon the skin of acne patients, and that I never use sulphur or other peeling agents. I have always treated acne as an internally caused disease and have employed creams and oils to cleanse the openings to the sebaceous glands.

For prophylaxis, I would rather use the oily, or the cream base form of medication, and, after rubbing it in, gently remove it with a soft cloth. I feel that my patient is better off because I believe that inflammation of the skin with medication only lengthens the duration of the acne.

Dr. Thomas W. Murrell, Richmond, Va.—The only advantage I can see in being old is that you can look back over a long time. If we follow Dr. Sutton's conclusions absolutely as far as acne is concerned, the x-ray is out. Now, I started in dermatology a very long time ago, and there were a great many years in which I had no x-ray in my office. If you take the masterpieces of the old literature, Crocker, Stellwagen, Hyde, you will find a great emphasis laid on fat intake, and we older men—I think Dr. Gaines will vouch for that—emphasize cutting fats out of the diet of these young people.

Along in 1919 or 1920, some of my patients in Richmond started to leave me and go to Baltimore or Philadelphia or Washington and get x-ray treatments from Ketron and others who were using x-ray, following MacKee's ideas. The improvements in these cases were so dramatic that I felt if I was to stay in dermatology I had to go into x-ray. I had tried x-ray around 1910 and quit it, but around 1921 I installed x-ray in my office a second time. Now if I compare the period in dermatology, say, from 1910 to 1925, with the fifteen years from 1925 to 1940, I am sure that the results that I am getting in my acne cases are far superior in the last fifteen years. I am very confident that that is so.

We hear in these sections many scientific presentations. We have contributions upon allergy. We have all had encyclopedic reports upon a patient by an allergist who obviously proves something from a scientific angle, but there is an enormous impracticality in carrying out the suggestions, and I believe that the failure in an enormous number of cases is not due to the lack of truth of this contribution but rather to the lack of ability to put it over. During thirty years of therapy I have treated acne with diet over a period and over another period I have relied upon sulphur and x-ray, and in contradiction to Dick Sutton, Jr., I still feel that in the latter period of my life, where x-ray was used, my results have been much better.

Dr. Toulmin Gaines, Mobile, Ala.—I agree with all the last speaker has said about the x-ray. I started my work coincident with the x-ray, so I cannot tell whether my work with acne is better with or without the x-ray, because I used it from the start, but I think I have been very successful in the treatment of acne vulgaris.

Dr. Sutton believes that the condition is caused by fatty diet, with which I agree. I do not depend on the x-ray. I depend on diet and x-ray and local methods of cleanliness.

According to my theory of acne, there is a keratosis of the sebaceous follicle, caused by an anomalous condition at the age of puberty. The sebaceous follicle takes on a keratotic condition which blocks it. When the patient uses too much grease and fat they accumulate in the sebaceous gland. The sebaceous gland, you know, is not a gland. There is exfoliation within this pocket, and there is a condition of fatty infiltration. These broken-down cells are shelled off with their fatty deposit and that is what causes the sebaceous secretion. It is not really a secretion; it is a fatty infiltration. So the use of fats certainly should be prohibited at the age of puberty. We prohibit sugars and fats. I do not let the patients eat chocolate candy, above all things.

Now, there has been a recent article in the *Archives* with an approach from the histopathologic standpoint. The authors found that there was a keratosis of these follicles very often, which begins at puberty. That would not explain, of course, the condition in a girl, but many anomalous sexual conditions occur in young people. I believe we are going to overcome the condition by the use of a hormone or vitamin, perhaps vitamin A, by the restriction of the fatty diets, and by local means, such as x-ray and removal of comedones, so the sebaceous glands will be free to remove the fatty secretion.

Dr. Martin F. Engman, Jr., St. Louis, Mo.—Acne is a disease which affects every human being who reaches puberty. Bloch showed this in his statistics. He also mentions that at about 18 years of age acne vulgaris attains its greatest degree of severity.

Our present treatment of acne is not very satisfactory. Relapses are the rule rather than the exception and are very discouraging both to the patient and to the physician. More research into the etiology of this disease may some time improve our therapeutic regimes.

I cannot entirely agree with the essayist in his hypothesis as to the etiology of acne. I think there are at least three basic factors concerned in the production of acne, namely: the hereditary characteristics of the individual, the activity of the sexual hormones, and the bacterial flora of the follicle. Our everyday clinical experience clearly indicates that the oily skin with its large follicles, which is the basis of acne, is inherited. The hormonal factor is less understood. At puberty the sex hormones become active and secondary sexual characteristics begin to appear. Adult distribution of hair begins. Apparently the follicles are activated, especially in the seborrheic areas, and acne simplex to a greater or lesser degree occurs.

Research into the nature and normal or abnormal function of the sex hormone is difficult because a normal base line has never been established. What is normal? During the acne ages the individual is young and emotionally unstable. Production of sex hormones may vary with the emotional stimulation and with contacts with the opposite sex. The whole sex hormone problem is relatively unknown at present.

The third factor in acne vulgaris is the flora of the follicle, particularly the acne bacillus. The fact that the acne bacillus is found in normal skin is not valid evidence that it is not pathogenic. Many pathogenic organisms may live in skin or other tissues and not produce disease until conditions are suitable for growth. At puberty the skin changes markedly. The acne bacillus finds a different culture medium. One finds this bacillus in the giant cells of acne lesions and in the walls of the cysts. Dr. Welch remarked to Engman, Sr., after examining such histologic sections, that he thought the acne bacillus had as much relationship to the lesion of acne as did the tubercle bacillus to the tubercle.

Dr. Herbert S. Alden, Atlanta, Ga.—If we are to believe what has been said and what we have read, then we are obliged, I think, to consider that both puberty and heredity are inevitable. I agree with Dr. Engman that the acne patient is unwise in his ancestors and unwise that he has to go through puberty. We have nothing we can do about that. We only have to cope with the situation. As I see it, too, seborrhea is also inevitable in puberty. If seborrhea is inevitable, and as we see it again the comedone associated with seborrhea is inevitable, then our acne, as we know it, is a situation that occurs and is not a disease. It then becomes a mechanical condition.

Now, I have felt, in my observation of the acne patient, that I have done better with the treatment of acne as a mechanical difficulty to be coped with rather than as a disease caused by any single or double set of factors. Hence, I have always felt that mechanical treatment was the best course to follow.

In my clinic work at Grady Hospital, in Atlanta, I have no x-ray for treatment. In the office, I have. Thus, I have a fair check on the local versus the x-ray treatment of these patients. In the clinic patient I

believe in time I get about as good results with the use of scrubbing with soap and water and lotions like lotio alba as I do in the office with my x-ray treatment and the use of lotio alba also. The only difference lies in the fact that the use of the x-ray shortens the course and gets the result quicker with less scarring.

I have never been an advocate of any strict dietary regime for reasons that seem obvious to me, first, because, as I say again, I look upon the disease as a condition based on mechanical factors; and second, because I have never had any success in impressing the patient with the use of the strict diet adhered to over a sufficient period of time to be of any real value.

However, I think the theory of the distorted fat and lipoid metabolism, suggested by Dr. Sutton, is very intriguing. I hope I can use his diets practically, but I still think that with the meticulous direction as to how the patient is to take care of it at home, the instruction that he is dealing with a condition that he is going to have to some extent throughout puberty, informing him we are not curing a disease but we are coping with a condition with his help and his continued treatment and care, we will usually have a happy result.

Dr. Dudley C. Smith, University, Va.—About two years ago a young girl on the medical service, who had been admitted to the hospital for complete study, was seen in consultation because of acne of the face. A part of her general examination included basal metabolism determination. This was found to be low (minus 19 per cent). This young girl was given routine instruction in regard to diet, local application, and so on. In addition the internist prescribed thyroid extract. Very promptly this young lady's acne improved and shortly disappeared completely. The result was so spectacular that I started doing routine basal metabolism determination on all cases of acne.

Since then almost all acne patients have been observed to have low metabolic rates, usually from minus 10 to minus 40 per cent. The occasional exception was probably not a true acne vulgaris, as suggested in Dr. Van Studdiford's discussion. I am convinced that the administration of thyroid extract with a low-fat diet, in addition to the usual routine procedures gives better results in acne vulgaris than any other plan of treatment. In practice I have not given thyroid extract without previous basal metabolism determinations, but I am sure that this can be done without hazard and with benefit. The basal metabolism determination is a rough guide for dosage.

The most important indication for the need of thyroid extract in patients is their ability to take this agent without untoward symptoms. The small dosage of thyroid extract frequently given has been the reason for the poor results with its use. The average case of post-adolescent acne can take 3-6 grains of thyroid extract (American standard) without untoward symptoms. In this connection, from a practical angle, it is best not to discuss with the patient the symptoms that might arise following thyroid intoxication. If a patient is told to watch his pulse and that he may have insomnia, the suggestion may cause these. The patient is seen at fairly frequent intervals, that is, about once a week at first and every two or three weeks later. Marked change in weight is considered a significant indication for changing dosage.

I am certain that Dr. Sutton's hypothesis in regard to the etiology and treatment on this basis gives a quicker and better result than any other method of managing acne vulgaris.

Local applications, I believe, have real value. A fat and resin solvent like a saturated alcoholic (95 per cent) solution with an addition of 2 per cent salicylic acid is a favorite application.

I should like to hear a discussion of the part resin of the wax in the sebum plays in the production of comedones and acne.

Dr. Harry M. Robinson, Baltimore, Md.—May I ask Dr. Sutton what he does for a patient who has both acne and pulmonary tuberculosis? Those specialists in pulmonary tuberculosis who have sent me patients with acne vulgaris for treatment have forbidden the use of restrictive diets, especially forbidding of milk, and have also forbidden the administration of thyroxin.

Dr. J. Lewis Pipkin, San Antonio, Tex.—Dr. Sutton has made a very distinct contribution in handling some selected cases of acne. We feel our cases do better under this regime.

We are not quite so heroic as he in regard to the administration of thyroid extract. We often go up as high as three or four grains a day, and in the majority of instances we feel that is adequate. As a general rule, the basal metabolic rate does not reflect definitely whether the patient will tolerate the thyroid or not. Probably the symptoms will better guide you in that respect.

Dr. D. Truett Gandy, Houston, Tex.—In regard to Dr. Sutton's use of thyroid it occurs to me to ask what happens when his patients stop taking it? Surely he does not keep them on thyroid indefinitely, and when it is discontinued do they not relapse, just as they do with other methods of treatment?

Dr. Robert Brandt, Augusta, Ga.—The frequency of acne is very different in different places. For instance, in Augusta I have seen a higher percentage of acne than ever before in my life. Up in North Carolina in the mountains and also here there are fewer cases, just looking at the women as you walk along the street. I do not know what the difference is. I thought it might be something in the diet, for instance, a diet very highly seasoned with a high amount of salt. If you can reduce the amount of salt you get an improvement of acne. But I am not sure that this is the only reason.

I want to ask if anyone knows why it is that in two areas close together the frequency of acne is often very different.

Dr. Andrew L. Glaze, Birmingham, Ala.—Dr. Sutton referred to giving the patient a quart of milk and finding that it made the acne worse. I think all of us will agree to that. Milk is one of the most important sources of iodine in the dietary. It is possible that a patient who has a hair-trigger sensitivity to iodine might in some instances have his acne made worse by the increased iodine intake.

Dr. Sutton asked how you can tell what patients are going to have acne and what patients will not. I agree with Dr. Engman's thesis that all have acne at some time or other. It is merely a question of degree.

An examination of any young woman between the ages of 11, 16, and earlier, for that matter, will disclose that in the favorite sebaceous areas of activity, on the chin, the glabella, the temporal areas and the nose,

there are patulous pores and more or less marked evidence of sebaceous activity, and in those individuals who do not develop the bad, frank picture of acne, one may prevent or tend to prevent those sequelae which, though mild, are still undesirable.

Those who are not specialists, and perhaps some of us who are, can better realize that there is no real conflict, only an apparent conflict, in all of the discussions that have appeared here today. There are different approaches and phases of the etiology. But we may go back to the primary factor, that embryologically oil glands are not set off in one place and follicles set off in another, but that a little invagination takes place in the fourth month and forms a little oil gland intimately connected with a follicle. In this holocrine gland, the sebaceous gland, nature apparently has designed a rather imperfect apparatus. The oil produced by the gland, as we all remember from our school days, is produced by the glandular cell's swelling up with oil and exploding and destroying itself. So the discharge from any normal oil gland is a mixture of the sebaceous oil and cellular débris. This in turn means, as we will remember, that we have a basal layer in the gland and in the duct, for that matter, of cells under the forced necessity of constantly and rapidly reproducing themselves in order to make up for the loss of destroyed cells. Of course we know this explains why there is a relative response to x-ray treatment in doses that the skin withstands very well.

Now, just why fat intake produces some perhaps not understood change in the character of the sebum produced by these cells when they swell up, burst and destroy themselves in order to discharge their fat, remains to be explained if faulty fat intake or faulty fat ingestion does interfere in some way with the normal pattern of oil production, either intracellular or after it is discharged into the duct. We can see with this basis that all of the discussions of treatment and prevention that have taken place here today deal with attempts to prevent the disorder, the disorganization, that results in the production of oil and in nature's attempt to pour it out on the skin to produce normal lubrication of the skin.

Dr. Sutton (*closing*).—Almost all of the sebaceous glands subject to acne (those on the face in contrast to those on the scalp which curiously do not develop acne, excepting those on the temples and the nape of the neck) can be kept from secreting by using local applications, at least to some extent. However, you can also get at the function of the sebaceous gland from the inside.

If you do nothing but express comedones, you do not make fat glands smaller, just as you do not dry up a cow by milking her.

Dr. Van Studdiford would call the severely deformed patient whose picture I showed tuberculous. I ask for proof that such cases are tuberculosis. This one developed from what looked like ordinary acne, during which the patient received an injection of iodized oil into his bronchiectatic lungs and immediately exploded with this inflammatory eruption. He responded favorably to my treatment. Histologic similarities between tuberculosis and acne are notorious and obvious, but histologic similarities do not define etiologic identities, though they may indicate similarities with regard to the chemical substances involved. It is the waxy fraction of the tubercle bacillus which evokes foreign body giant cells. It is the protein water-soluble fraction which evokes necrosis and polymorphonuclear leukocytosis. Studies of tissue reactions to non-living chemical fractions of oils of various kinds have led to recognition of the varieties of inflammatory response that can occur. Only a limited assortment of inflammatory responses is possible, no matter how various the chemical instigators of them are. Those deep cysts in the deep-seated "tuberculoid" acne are sterile. They do not even have the acne bacillus in them.

I ask you to take any acne patient and add to his diet a quart of milk a day, and see what happens. He will get worse invariably, just as surely as if you add iodide to his diet without counterbalancing it somehow. You can allow iodized salt to acne patients if thyroxin is given, too.

Inheritance of the tendency to acne is important. Look at your family with four children; we will say two of the children have acne and two do not, while one parent has had acne and the other has not. A decided physical resemblance between the acneic children and the acneic parent will be obvious. People inherit their chemical patterns, I believe, as they inherit the shape of their nose and other physical features.

Dr. Van Studdiford suggested that thyroid function is elevated in the summertime. I do not know that that is so. Perhaps there is less utilization of thyroid secretion during the summer, when a person is not obliged to burn his oil to keep himself warm. Thyroxin is used in metabolizing oil, just as insulin is used in metabolizing dextrose. Furthermore, in the summertime the intake of vitamin A is increased. Vitamin A is antagonistic to and neutralizes thyroxin. This is news to many of you. George Andrews and I had quite a debate about that, but experiments have proved it. You can kill a guinea pig with huge doses of vitamin A, and the guinea pig dies with all the evidences of acute complete hypothyroidism. He loses weight, his hair falls out, he weakens and dies.

This antagonism is a fundamental fact in considering the etiology of acne. A B D capsules and vitamin concentrates nowadays are popular. I believe they make acne worse because of their vitamin A content. That may be contrary to the oninions of some of you.

Knowledge of what is fatty is important in designing a diet, for the diet is a fat blockade, and if it leaks it does not work. You would think that soda crackers, made mainly from wheat which is a plant substance and is not oily, would be allowable, but an ordinary soda cracker is 10 per cent oil by weight. Potato, as it comes from mother earth, is one-tenth of one per cent oil by weight. Split it and fry it, and it takes up 30 to 40 per cent oil by weight. Some meats are much more oily than others. The pig is very oily. Pork, ham, bacon, and sausage must be excluded. Lean beef is 10 per cent oil. I did not detail the diet in speaking to you because I limited myself to twenty minutes. One cannot allow much meat.

There are some cases reluctant to respond to this therapy. There are plenty of things that no one knows about fat metabolism. Some 20 per cent of my patients may eventually require x-ray therapy, simply because I cannot stop their fat secretion by the diet they follow and by thyroxin, while x-ray does stop their fat secretion by disabling the glands. No one claims that x-ray helps acne in any other way, and all agree it is a symptomatic, not an etiologic, therapeutic effort.

Dr. Gaines mentioned keratosis at the sebaceous follicle orifice in association with the comedo. That is

recognized as the answer to the question, "What do we see anatomically?" But it makes no attempt to answer the question, "Why?" Not only is the gland a structure which exfoliates within a pocket, as Dr. Gaines neatly put it; the whole epidermis exfoliates, and is greasy in acne. You cannot modify the greasiness of the entire epidermis by x-ray therapy alone, other than temporarily, for a patient who shows x-ray atrophy of the skin may still be greasy; and from time to time the little islands of fat which you can feel with your fingertip still within his skin will provoke foreign body inflammation.

Many disturbances are due, not to lack of nutrient substances, but to oversupply. You can make a person thin, anemic, tired and weak by overdosing with vitamin A. You can make a face break out by overdosing with milk and cream. Many a thin girl feels worse and her face becomes much worse when she tries to gain weight by drinking chocolate malted milks. If you put her on a low-fat diet and give suitable dosage of thyroxin, she will lose her nervousness, sleep better, eat voraciously, feel fine, and gain weight.

Part of the inherited basis of acne, which Dr. Engman mentioned, might be due to "inheritance" of dietary habits. Dietary habits are pretty well fixed things. One family eats a greasy diet, and another family does not. You may recall Herman Goodman's study of the incidence of acne among Jewish people. Jewesses suffered from acne far more commonly than his control population, simply, I believe, because of the oiliness of their food.

Not even in puberty is an individual nothing but sex glands. There is more to an individual than those alone. It is known through experimentation and quantitative chemical studies that thyroxin and anterior pituitary secretion will lower the blood fat level. Pituitary lowers lipemia in six hours. Thyroxin takes longer, but it certainly lowers it in every individual. An overdose conspicuously produces headache in my experience. Any additional thyroxin is too much for a person whose supply is complete and normal, whose "bucket is full"; but an individual whose "bucket" of thyroid secretion is leaky and empty requires a large dose to refill and then a small dose to maintain. On low-fat diet the requirement for thyroxin is considerably diminished and you can keep your patient filled with a moderate dosage.

Dr. Gandy asks whether one should continue thyroxin dosage indefinitely. I should say you can give thyroid extract forever, provided it is given in suitable dosage, just as you can give insulin forever and your patient is the better for it, if you dose it suitably. The suitable dosage of thyroxin must be determined by building up to an overdose, then dropping to the tolerated dose.

Others have seen that thyroid has helped acne patients. I have found reports back as far as 1902, at any rate. But thyroid extract alone is not enough, just as in diabetes mellitus insulin alone is not enough. You must take the diet into consideration at the same time.

Dr. Robinson presented the problem of acne complicating tuberculosis. I really do not know how to handle that. I doubt that you would damage a tuberculous individual by tentatively putting him on a comparatively low fat diet. There is apparently some relationship between foreign body giant cell reaction to tubercle bacilli, the deposition of oily stuff around the bacteria, and immunity to tuberculosis. I should handle such a case with great care, and perhaps allow him to keep his acne as the less of two evils.

The adequate dose of thyroxin must be determined clinically. You must push it until symptoms of hyperthyroidism appear. Only then can you know that the bucket has been filled, and after that you must drop the dose to the tolerated level.

People differ in their dietary habits, Dr. Brandt, and geographic regions differ in the thyroid activity of their inhabitants. Goiter belts are familiar. There must be racial differences, too. The Eskimo on a high fat diet does not suffer from acne conspicuously.

You will note that the high fat diet is a diet rich in animal substances. The human being, with his biologic relationship to the vegetarian ape, perhaps should be more vegetarian than he is.

LARYNGOTRACHEOBRONCHITIS*

By VAN DYKE HAGAMAN, M.D.
Jackson, Mississippi

Laryngotracheobronchitis is an inflammatory disease of the respiratory system caused by pyogenic organisms and it is usually fatal. In part, the diagnosis and much of the treatment, such as the use of bactericidal drugs and general care, are pediatric problems, but the otolaryngologist, especially if he is interested in endoscopy, is frequently consulted because of the alarming obstructive phenomena that occur in the course of this disease.

The purpose of this paper is to report three fatal cases of laryngotracheobronchitis and to utilize the observations of the laryngologist concerning the diagnosis, course and treatment of this disease.

REPORT OF CASES

Case 1.—B. R. G., a white girl, aged 9 months, was admitted to the Mississippi Baptist Hospital December 16, 1939. For three days the child had been treated at home with expectorants and sulfanilamide. The family physician had administered diphtheria toxoid three weeks prior to admission.

Physical examination revealed that the larynx was red and edematous. The general condition was alarming due to cyanosis and respiratory difficulty. The temperature was 101° F. Cultures from the pharynx and larynx taken with an ordinary throat swab were later reported negative for diphtheria and positive for staphylococcus.

It was obvious that respiratory obstruction was a major problem, and low tracheotomy was performed the day of admission. The tracheotomy by itself relieved symptoms of respiratory distress, and thick mucus and pus were obtained by suction from the lower trachea and primary bronchi. Following removal of this ma-

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*Read in Section on Ophthalmology and Otolaryngology, Southern Medical Association, Thirty-Fourth Annual Meeting, Louisville, Kentucky, November 12-15, 1940.

terial by aspiration, there was prompt clinical improvement, which lasted until similar secretions reaccumulated, and aspiration had to be repeated. On the third day several bronchoscopies were necessary for removal of dried crusts from both bronchi. On the third night of admission the patient suffered a dramatic physical collapse, accompanied by physical signs of spontaneous left pneumothorax with shift of the mediastinum to the right. Death occurred the following morning. At no time during the course of the disease did the temperature rise above 103° F.

Necropsy.—The anatomic diagnosis reported by the pathologist included, (1) pseudomembranous laryngotracheobronchitis, and (2) marked emphysema of the right lung with spontaneous pneumothorax of the left pleura. There was only slight evidence of bronchopneumonia in both lungs. In addition, emphysematous blebs were described under the surface of the visceral pleurae, and it was suggested that the pneumothorax probably resulted from spontaneous rupture of one of the blebs. The exact location of the leak into the pleural cavity was not demonstrated. Of major interest to the laryngologist was the location of the pseudomembrane with respect to the tracheotomy, because the primary bronchi and distal three centimeters of the trachea were lined by a fibrinopurulent pseudomembrane and, although these passages were anatomically patent, it was obvious that the inflammatory reaction in this location was responsible for the emphysema and resultant pneumothorax.

Case 2.—B. J. G., a girl, aged 10 months, was admitted to the Mississippi Baptist Hospital January 28, 1940. One day earlier the baby became ill with croup, fever and respiratory distress.

On physical examination the larynx was found to be red and edematous. Cyanosis, restlessness and difficulty in breathing were so pronounced that a low tracheotomy was immediately performed. Cultures from the larynx and pharynx were negative for diphtheria. Actual respiratory stridor was relieved by the tracheotomy, but cyanosis, restlessness and rapid respiration continued. Sulfapyridine was administered in addition to routine medical care. On the third day of hospitalization, an x-ray picture of the chest demonstrated a pneumothorax on the right side and the child died early the following morning.

Necropsy.—The report of the pathologist described the pneumothorax on the right, emphysematous blebs on the pleurae, and a slight amount of bronchopneumonia. In the reports this case was similar to *Case 1.* However, in this case the inflammatory areas were found to be proximal to the tracheotomy, and no obstructive inflammation of the respiratory passages was demonstrated distal to the opening. The pathologist also took cultures of the larynx, but they were subsequently reported negative for diphtheria.

These two cases demonstrate the importance of spontaneous pneumothorax as a complication of laryngotracheobronchitis. In the first case, the onset of pneumothorax was sudden and dramatic and evidently caused death in a child whose condition had been previously serious but not alarming. In the second case, the pneumothorax was overlooked clinically until the baby was nearly dead. In view of the alarming respiratory distress that was in evidence throughout the course of the illness in this case, and in view of the fact that the tracheotomy opening was placed distal to the obstruction, it is not unreasonable to suspect that the pneumothorax was present on admission and gradually increased in extent.

The use of sulfanilamide in one case and sulfapyridine in another failed to influence the course of either favorably, but in both cases the problems involved were mechanical rather than septic and related to the development of pneumothorax. Sulfathiazole was not available at the time these cases were encountered.

As is customary in all such cases the laryngologist worked in collaboration with the pediatrician.

The next case is reported briefly to demonstrate that the laryngologist has a diagnostic problem as well as a problem of mechanical therapy in cases of laryngotracheobronchitis.

Case 3.—L. A. W., a white girl, aged 3 years, was admitted to the State Charity Hospital at 5:00 p. m. May 3, 1940. For several days the child had been treated at home for bronchitis and sulfapyridine had been administered. The patient was admitted to the hospital in a moribund condition. Respiratory difficulty was obviously the major problem.

Thick pus was removed endoscopically from the lower respiratory passages. The larynx and pharynx were inflamed, but there was no pseudomembrane in the upper respiratory passages. Cultures of the larynx and pharynx for diphtheria were taken. Bronchoscopy revealed a suspicious membrane with purulent secretion and obstruction in the trachea and bronchi. Diphtheria antitoxin was administered to this child because of the bronchoscopic findings, but death occurred thirteen hours following admission. There was no opportunity for further clinical investigation, and necropsy was refused. When cultures were later reported, those taken from the larynx and pharynx were negative for diphtheria, but those taken from the pseudomembrane of the trachea and bronchi were positive.

This case illustrates the importance of cultures from the actual pseudomembrane in all cases of diphtheritic inflammation to demonstrate the presence of diphtheria organisms. It is not implied here that all cases of laryngotracheobronchitis are diphtheria, but this case would have been included with those caused by pyogenic organisms if reliance had been placed upon cultures taken from the pharynx and larynx alone. In fact, since cultures were never made from the actual inflammatory areas in the lower respiratory passages of *Case 1,* we might have been dealing with a diphtheritic inflammation in that case, too, even though diphtheria toxoid

was given three weeks before admission to the hospital. Why would it not be possible to have a mixed infection? A case in 1931 had a positive diphtheria culture from the pharynx and a streptococcic septicemia. A negative conclusion as regards diphtheria is very important and is justified only after six to eight days have elapsed from the onset of symptoms. This idea was substantiated by a hospitalized case in our locality last winter. The diagnosis was laryngotracheobronchitis and the case required intubation. The patient did not recover until diphtheria antitoxin was given on the sixth hospital day, at which time the culture was positive for diphtheria. Repeated cultures had before this time all been negative for diphtheria. In Mississippi diphtheria is endemic, and we now urge the general practitioners to give diphtheria antitoxin to patients with laryngotracheobronchitis early in the course of the disease.

CONCLUSIONS

We look to the use of bactericidal drugs and improvements in apparatus and methods of general care such as special types of humidifiers to help us in the treatment of laryngotracheobronchitis during the coming winter. In addition to these measures, however, the laryngologist has a profound responsibility in these cases. It is his job to keep open the respiratory passages in so far as possible by tracheotomy and removal of purulent secretions and by bronchoscopic removal of lower airway obstructions. In two of the three cases reported, the laryngologist was defeated by the development of spontaneous pneumothorax. The laryngologist also has a diagnostic and epidemiologic problem in ruling out diphtheria when the inflammatory areas in cases of laryngotracheobronchitis can be cultured only through the bronchoscope and not by direct swabbing of the throat. This latter responsibility was demonstrated in *Case 3* of this series.

There has been no attempt in this paper to review the literature. While most of the ideas are our own, we acknowledge to borrowing extensively from Richards, Davison, Evans, Gittens, Jackson, Baum, Simpson, Holinger, and Brenneman.

DISCUSSION (*Abstract*)

Dr. W. Likely Simpson, Memphis, Tenn.—Dr. Hagaman's case reports emphasize the unfavorable prognosis in many of these little patients.

The routine procedures which are carried out by the University of Tennessee group at the Baptist Memorial and John Gaston Hospitals in Memphis are as follows: when these little patients are brought into the hospital, a culture is made through the direct laryngoscope. If the larynx has the appearance of diphtheria, diphtheritic antitoxin is given at once. If the larynx and trachea have the appearance of the ordinary laryngotracheobronchitis, general treatment only is carried out. If breathing is difficult, a tracheotomy is done. Usually a small bronchoscope is inserted while this is being done. The room is kept warm and a humidifier is installed in the room. The child is encouraged to take fluids. If for any reason fluids are not taken by mouth, intravenous glucose, and so on, are given.

Before the type of bacteria is determined, some form of chemotherapy is usually carried out and is changed according to the laboratory report.

It is understood that no sedatives such as paregoric and no drugs such as atropine are to be given at any time.

Suction with the rubber catheter is carried out through the tracheotomy tube. If the child becomes quite restless and the tracheotomy tube is open, a careful bronchoscopy with a 3.5 millimeter lumen tube is carried out.

The group at the University believes that quite a high percentage of the patients are saved through the removal of tenacious mucus and crusts in the trachea and main bronchi through the bronchoscope. By means of a suction and forceps the carefully done bronchoscop, does little if any harm, and usually none, and is a means of saving lives. The restless child with laryngotracheobronchitis usually suggests a bronchoscopy. Some of the more obstinate cases need a bronchoscopy every one or two hours. Fifteen to 20 bronchoscopies are not unusual. A pneumothorax complicates this condition, but some of our cases, even though pneumothorax with crusts is present, with the aid of the bronchoscope get well.

Dr. Murdock Equen, Atlanta, Ga.—This most distressing condition is usually seen in very young children and its seriousness is in proportion to the age of the individual. This fact is easily understood when one considers the size of the tracheobronchial tree in young and older individuals and that an equal amount of swelling to one individual may mean extreme dyspnea, which is always progressive, and to another a mild discomfort in breathing.

The value of an early tracheotomy before the heart wears itself out, the keeping up of body fluids by saline and transfusions, and the administration of chemical therapy to a high blood concentration is well known. Rest is most important and frequent examinations tend to make young children cry and fret, thus expending their energy.

By keeping these children in a room saturated with moisture there will be less tendency for scab formation. The instillation of various solutions through the tracheotomy tube and the use of a catheter with negative pressure have frequently been suggested, but the less intratracheal manipulations the better it is for the patient.

It is to be hoped that this paper will be widely read by both the general practitioner and the pediatrician, as they are the ones who first see these patients.

Dr. L. Chester McHenry, Oklahoma City, Okla.—I am very much interested in the problem Dr. Hagaman has brought before us and wish to compliment him upon

presenting us with fatalities. These are not at all necessarily bad results. Fatalities occur in spite of everything we can do and I think that all of us who see these cases know that we are going to lose a fairly large per cent of them.

The cultures, taken through the laryngoscope as described by Dr. Simpson, showed staphylococcus in some half dozen cases seen last year. I had been under the impression that most of these severe cases were caused by the streptococcus. In approximately half of these cases we were able to remove a large dried mass of secretion from the subglottic region and obtain relief without performing tracheotomy. The after-care of these cases is practically the same whether they have been tracheotomized or not. We use the croup tent routinely. Dr. Equen's idea of a room where the humidity may be kept very high without raising the temperature so much sounds very excellent for this purpose. If the inspired air is not kept moist and the intake of fluids kept high, sticky secretions collect and cause recurring obstruction.

Chemotherapy has been of great assistance in these cases.

Dr. Simpson's routine method of handling such cases is very efficacious and I shall add some of his points to my own procedures.

Dr. Fitz-Hugh, in discusing tracheotomies yesterday, mentioned pneumothorax as a fairly frequent complication. I wonder if we do not have pneumothorax more often than we realize. Probably some of those who develop subcutaneous emphysema of the neck have pneumothorax also. It is probably not discovered because it does not seriously complicate the picture. In his cases it apparently caused no trouble.

Dr. Hagaman (closing).—A personal communication from Dr. Richards recently was to the effect that the special humidifier, which is put out by the A. C. Gilbert Company of New Haven, Connecticut, is one of the newer adjuncts in the treatment of the disease. Davidson had reported the humidifier earlier in the year. This is the humidifier which produces about a hundred per cent saturation without increasing the temperature over 70°. Dr. Richards said in addition that he thought sulfapyridine was definitely giving better results.

Dr. Holinger, in a letter, reported a large reduction of cases during the past twelve months. He also questioned in some few cases the good results of sulfanilamide and sulfapyridine. He felt the increase in cyanosis was harmful.

I have avoided discussing the symptomatology and treatment of this disease. The literature is really filled with excellent material.

GIRL BORN WITHOUT LEGS AND WITHOUT ARMS: REPORT OF A CASE*

By JULIUS L. LEVY, M.D.
Clarksdale, Mississippi

Following is a case report of an infant delivered by breech presentation at full term, with complete absence of the four extremities at birth. In the author's review of the literature, no similar case was reported.

The author was not consulted until the day of the delivery. Labor progressed normally, and the condition of the mother was good. A stillborn female infant weighing 5 lbs. 8 oz. was delivered by breech extraction February 14, 1941. Complete absence of all extremities was observed. The x-ray and picture of the infant were taken a half hour after birth.

The infant's head and trunk are, in appearance and measurement, much the same as those of a normal child at birth; but the infant is entirely destitute of upper and lower extremities. Her entire height is 20½ inches. There is no curvature or deformity of the spine, but the sacral vertebrae are perhaps a little less incurved, somewhat more rounded and prominent, than usual; the chest is well developed and of fair size and form, and measures 20 inches in circumference at the nipples. The pectoral muscles are well developed.

The skin at the end of the shoulders is smooth and presents no mark or indentation where the arms should come off.

There are no lower extremities; but in the position of the head of each femur there is a small projecting mass which measures half an inch in length and ¾ inch in diameter. Each of these projections is attached to the trunk by a peduncle of about half its own diameter; but the peduncle is so short as, on first examination, to be scarcely perceptible. These small masses are quite soft and appear to consist only of skin, cellular tissue and fat. There is no trace of either femur. In the position of the acetabulum a slight ridge may be seen, which probably corresponds with the margin of the cavity which ought to exist.

The father and mother are of Greek descent, ages 27 and 38. There were no malformations in either of their families. They had been married for two years and this was the first pregnancy.

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M. Y. DABNEY, M.D., Editor
C. P. LORANZ, Secretary, Treasurer and General Manager

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EDITORIAL DEPARTMENT

SOUTHERN MEDICAL ASSOCIATION
Thirty-Fifth Annual Meeting
St. Louis, Missouri, November 10-13, 1941

ST. LOUIS MEETING

PROGRAM

A review of the unusually strong program now nearing completion for the thirty-fifth annual meeting of the Southern Medical Association promises something of interest for every attending physician. The clinical sessions by St. Louis men beginning on Monday afternoon, November 10, and lasting until Tuesday noon, the nineteen section meetings from Tuesday noon until Thursday, the closing day, and the scientific and technical exhibits in addition to the entertainment features will form a veritable scientific power house.

SCIENTIFIC EXHIBITS

Particularly impressive is the large number of applications for space for exhibits which are in the Association's office at the present time. The exhibit program this year promises to exceed any other in the history of the Association and an unexcelled variety representing all phases of medicine is to be found. Physicians who wish to participate with a scientific exhibit at the St. Louis meeting should communicate at once with the Association's office and secure an application. Only a limited amount of space is available at present.

HOBBY EXHIBITS

As a follow-up to the Hobby Exhibit inaugurated at the Louisville meeting last year, the Southern Medical Association will again this year sponsor an exhibit depicting hobbies of physicians. Those interested in displaying paintings, pen and pencil sketches, photography, stamps and coin collections, sculpture, and so on should apply to the Association's office in Birmingham.

HOTELS

Every physician attending the St. Louis meeting may be assured hotel accommodations to his liking both as to quality of rooms and price; for no city in Southern Medical territory is better qualified than St. Louis to handle a group comarable in membership to the Southern Medical Association.

Only the Jefferson, official headquarters hotel, has accepted reservations for the entire number of rooms which will be available during the meeting, and is completely filled. Other good downtown hotels such as the Statler, DeSoto, Mayfair, Mark Twain, Lennox, Claridge, American, Majestic and Warwich have ample rooms at reasonable rates. All downtown hotels are only a short walk from the Municipal Auditorium where all scientific activities are being held.

Uptown hotels are particularly suited for those who drive to the meeting in their own automobiles. Among the better uptown hotels only from ten to twenty minutes ride from the Auditorium by car are the Coronado, Melbourne, Park Plaza, Chase, Kings-Way, Gatesworth, Fairgrounds, Forest Park and Roosevelt.

Even with the abundance of hotel rooms afforded by St. Louis, the more desirable accommodations will go to those who make reservations in advance. Those who have not yet obtained rooms should write today outlining their requirements to Hotel Committee, Southern Medical Association, Dr. J. Hoy Sanford, Chairman, 910 Syndicate Trust Building, St. Louis, Missouri.

MINERAL OIL IN PREGNANCY

Constipation is a subject which has occupied the attention of physicians over the centuries, though one sees fewer medical papers upon the subject than formerly. Mineral oil as a non-habit-forming laxative was presented to the public early in this century, and became overwhelmingly popular. It is frequently advocated as a

routine in pregnancy. It has been said to have absolutely no physiologic action, merely to lubricate the intestine and speed the passage and excretion of fecal matter. Whether it is wholly inert in the body is now being questioned. Within recent years it has been reported that it absorbs the fat-soluble vitamin A, which then passes outside the intestinal tract in mineral oil solution, instead of being digested and metabolized with other food materials. This has been commented upon particularly in treatment of tuberculous persons, who are usually given large quantities of vitamin A.

The effect of mineral oil upon utilization of vitamin K, the vitamin necessary for blood clotting, has been investigated by workers from Cornell University Medical College. The prothrombin content of the blood is believed to vary directly with the vitamin K intake. Maintenance of a normal prothrombin level in the mother's blood is advised for prevention of hemorrhagic disease of the newborn and it is also possibly of value in prevention of postpartum hemorrhage. Javert and Macri,[1] while making routine studies of the prothrombin content of the blood of a group of pregnant women, noted that the level sometimes fell suddenly in spite of the administration of abundant vitamin K. It was learned that in these cases the women were taking mineral oil. Prothrombin fell in a third of the patients when the oil was taken intermittently, and in more than two-thirds when it was taken daily. Hypoprothrombinemia thus commonly occurred in pregnant patients who took mineral oil. It was also observed in non-pregnant patients under the same conditions, during administration of vitamin K and mineral oil. It could be controlled by parenteral administration of vitamin K, which eliminated the necessity of absorption of the vitamin through the alimentary mucosa. The routine prescription of mineral oil, therefore these authors consider to have some deleterious effects hitherto unknown.

One must also conclude that merely the prescription by the physician and consumption by the patient of vitamins does not assure a physiologic supply. Other substances than mineral oil may interfere with vitamin utilization in the body, other drugs, or certain refractory physiologic states; and with different diets and different individual vitality, there are different quantitative requirements for vitamins, about which more will eventually be learned.

1. Javert, Carl T.; and Macri, Cesira: Prothrombin Concentration and Mineral Oil. Amer. Jour. Obst. & Gyn., **42**:409 (Sept.) 1941.

HYPERTENSION

The problem of high blood pressure has been investigated from many points of view and it would appear that there are numerous causes for this particular pathologic response. As Forbus[1] has said discussing the relation of Dorothy Reed cells to Hodgkin's disease and to brucellosis, the body possesses only a limited number of mechanisms of reaction to injurious agents, and it must repeat itself in its chemical and morphologic responses to totally different stimuli.

The organic response perhaps to several different stimuli is the formation of the Dorothy Reed cell. The organic response to another quite large and divergent group of stimuli is hypertension.

Doles[2] has shown that a protein-poor diet can be a factor in the production of elevated blood pressure and macrocytic anemia, and that persons with this type of hypertension have a deficient gastric juice.

For many years it was believed that increased arterial pressure was always accompanied by kidney disease. More recently, a group of cases of hypertension without demonstrable kidney disease was set aside in medical classification: the cases of so-called essential hypertension. A large amount of new investigative work has also recently gone into studies upon the type of hypertension which follows kidney disease. Goldblatt[3] and his associates, in 1934, showed that a rise of blood pressure regularly follows mechanical occlusion of the blood supply of one kidney of an experimental animal, even while the other kidney remains normal. A substance has been extracted from kidneys which raises the blood pressure of normal animals, and another which lowers it, and with the latter various attempts at therapy of clinical hypertension have been made.

Clinical surgical work has expanded, from the accumulated evidence that a single injured kidney may excrete a toxic substance which

1. Forbus, W. D.: Variations in Morphologic Reaction to Injuries. South. M. J., **32**:1208, 1939.

2. Doles, H. M.: Physiologic Factors Affecting Blood Pressure. Ibid. **31**:858, 1940.

3. Goldblatt, H.; Lynch, J.; Hanzal, R. F.; and Summerville, W. W.: Studies on Experimental Hypertension: 1. The Production of Persistent Elevation of Systolic Blood Pressure by Means of Renal Ischemia. J. Exper. Med., **59**:347, 1934.

causes hypertension and that this type of high blood pressure is relieved by nephrectomy. In these cases it must be known that one kidney caused the hypertension and that the other is functioning normally.

Schroeder and Steele,[4] of New York City, have observed certain cases in which hypertension developed after kidney stone. Hydronephrosis, they say, also was commonly followed by hypertension. One may understand that a kidney attempting to swell as the hydronephrotic one does, would be constricted by its surrounding tissues like the experimental normal animal kidney bound in a cellophane capsule, and thus would result a condition resembling the laboratory ones for interference with blood supply and production of kidney ischemia and hypertension.

Schroeder and Steele also note that an aberrant renal artery constricting the ureteropelvic junction may be a cause of hypertension. These are kidney-produced clinical hypertensions. And it is of interest to observe the types of kidney disease which may precede them.

According to animal work of Goldblatt, Kahn and Lewis,[5] occlusion of a ureter does not result in elevated blood pressure; only occlusion of the kidney blood supply does this. On the contrary, if the main artery of one kidney is constricted and the ureter occluded at the same time, if the opposite kidney is normal, the expected rise in blood pressure does not occur. If the normal kidney is then removed, the blood pressure rises. Ureteral occlusion in some way prevents either production or activity of the pressor substance of the ischemic kidney, if there is a functioning kidney on the opposite side.

One must conclude that a certain few clinical cases of hypertension with one diseased kidney poisoning the blood stream with pressor substances and with another normal kidney, may benefit by nephrectomy. Hypertension is not so completely in the field of the internist as has been thought. However, the selection of cases with one good and one bad kidney and no other possible cause for hypertension will be difficult for a long time. Dietary causes and the presence in the body of toxins from infectious diseases or focal infections remain prominent.

4. Schroeder, Henry A.; and Steele, J. M.: Studies on "Essential" Hypertension. Arch. Int. Med., **68**:261 (Aug.) 1941.

5. Goldblatt, H.; Kahn, J. R.; and Lewis, H. A.: Studies on Experimental Hypertension. Arch. Surg., **43**:327 (Sept.) 1941.

TWENTY-FIVE YEARS AGO

From Journals of 1916

Pernicious Anemia.[1]—Splenectomy seems to result in the greatest stimulation of the bone marrow. * * * However, it does not alter the essential course of the disease * * * from splenectomy one can obtain stimulation but once.

Pernicious Anemia.[2]—Splenectomy caused a quick and marked improvement in 64 per cent of all patients * * * a large number have failed to maintain this improvement. * * * It would appear to be not only justifiable but in many cases an advisable procedure.

Southern Medical Meeting in Atlanta.[3]—Officers: Editor, Dr. Seale Harris; Associate Editor, Dr. M. Y. Dabney; Business Manager, Mr. C. P. Loranz; President, Dr. Robert Wilson, Jr.; Section Chairmen: Medicine, Dr. K. H. Beall; Surgery, Dr. J. H. Blackburn; Public Health, Dr. W. S. Leathers; Eye, Ear, Nose and Throat, Dr. J. W. Jervey; Southern States Association of Railway Surgeons, Dr. Southgate Leigh. ¶The increase in membership from 600, in 1911, to more than 5,500 at this time, is not more phenomenal than the improvement in the scientific work at the meetings of the Southern Medical Association. * * * The Atlanta Meeting will mark the beginning of a new era in the work of medical societies in that it will be the first large assembly where a part of each day will be given over to clinics. Atlanta's immense auditorium, in which all of the general and section meetings of the Association will be held, is within three blocks of the Grady Hospital and the Atlanta Medical College buildings, which together can provide operating and clinical amphitheaters and lecture rooms that will comfortably seat two thousand physicians.

1. Lee, R. I.; Minot, George R.; and Vincent, B.: Splenectomy in Pernicious Anemia. J.A.M.A., **67**:719, Sept. 2, 1916.

2. Krumbhaar, Edward: Late Results of Splenectomy in Pernicious Anemia. Ibid., p. 723.

3. Editorial: The S.M.A. Meeting and Clinics. South. Med. J. **9**:939 (Oct.) 1916.

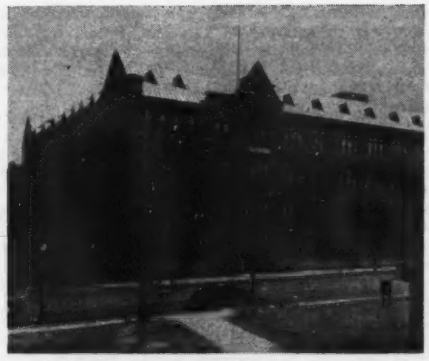
St. Louis—Where We Meet

ST. LOUIS AS A MEDICAL CENTER*

HISTORY OF ITS MEDICAL COLLEGES

During the first thirty-five years which followed the establishing of the trading post that was destined to become the City of St. Louis, a doctor arrived occasionally at the village but none remained long for one reason or another. There were no conveniences and life was too primitive for men of education and culture. Not until the year 1800 did the first physician arrive who was to have a permanent residence here. It was Dr. Antoine Francois Saugrain who had been educated at Paris, France, and who had practiced for a while at Gallipolis, Ohio. He enjoyed a splendid reputation among the best people of the town, had a large private practice and maintained a garden where he cultivated

*Prepared by Dr. Robert E. Schlueter, St. Louis, for the Sub-Committee on Publicity, Dr. Arthur W. Proetz, Chairman, of the General Committee for the St. Louis meeting.



Washington University School of Medicine.

medicinal plants for drugs to be used in his practice. He died in 1820 and there are quite a number of his descendants still living here, some of whom are physicians.

While other physicians arrived thereafter until there were several dozen very reputable doctors comprising the local medical profession, nothing startling happened for another thirty-five years. Then the already famous Dr. William Beaumont came to take charge of the United States Army Medical Supply Depot at the St. Louis Arsenal. Since his duties as an army surgeon did not require his entire time he had the privilege of engaging also in private practice, and soon had a large following among the people of St. Louis. Although he met with a cordial reception from some of his colleagues, his now well known experiments on digestion did not make him welcome with those who had a tendency to be envious. Nevertheless his presence seems to have stimulated the doctors to achieve greater things and the St. Louis Medical Society was organized in that same year, 1835. This organization also petitioned the St. Louis University to establish a medical department in the following year, thus founding the first medical college west of the Mississippi River. The available records of this school are so fragmentary that very little is now known of the activity of its faculty. At any rate it seems to have ceased to function by 1840.

At about the latter date Dr. Joseph Nash McDowell, nephew and one-time assistant of the great Ephraim McDowell of Kentucky, arrived and founded the Medical Department of Kemper College. It later became the Missouri Medical College, or "McDowell's College." In 1841 another group of medical men assembled mostly from other towns, organized another medical department of St. Louis University. On the whole this faculty was far more competent than those local teachers who had been chosen in 1836. Among them was the learned young Dr. Charles A. Pope, who later was in apparently full control of the school so that when it bore the name St. Louis Medical College and had no university connection it was popularly known as "Pope's College." There was also the brilliant Dr. Moses L. Linton, who in 1843 founded the first medical periodical in this western region and remained the editor of the *St. Louis Medical and Surgical Journal* until his death in 1871.

These two proprietary medical colleges continued to function well for more than half a century, despite a factional enmity between the two faculties, until medical science and education had progressed to a point where private ownership of a medical school was no longer profitable. The consultation fees of the professors were no longer adequate to compensate them for the loss of their financial investment. Other medical colleges were established here by ambitious doctors who were not favored by appointment as medical instructors so that there were at one time about one dozen such schools in the city, two homoepathic, two eclectic, and one giving only evening instruction. That was at the beginning of the last decade of the nineteenth century. From this array of privately owned medical schools which marked an epoch-making effort at medical education in the final years of the nineteen hundreds, only four have emerged.

The St. Louis Medical College became affiliated with Washington University in 1891 and in 1899 joined with the Missouri Medical College to become the Washington University School of Medicine. The first executive faculty was chosen from the two schools, an equal number taken from each school. Dr. Henry Hodgen Mudd was the first dean but this gentleman died after only about six months service in that capacity. The two medical college buildings with their large outpatient clinics were utilized for instruction, as were the wards and clinics of St. John's and St. Louis Mullanphy Hospitals which had been controlled by the colleges for many years previously. Soon, however, the comparatively new Missouri Medical College buildings at Jefferson and Lucas Avenues were remodeled into the Washington University Hospital and all non-clinical departments housed in the buildings of the St. Louis Medical College at 1812-1820 Locust Street. Thus the available facilities were employed until the new buildings at Euclid and Scott Avenue were finished in 1915 and the Barnes Hospital with the adjoining spacious outpatient department was erected. Since then the St. Louis Children's Hospital, the St. Louis Maternity Hospital, McMillan Eye, Ear, Nose and Throat Hospital, the Oscar Johnson Institute for Research in Ophthalmology and Oto-Laryngology, and the Edward Mallinckrodt Institute of Radiology have been added to furnish the highest degree of cooperation between the various departments.

In 1886, a group of enterprising local physicians and

St. Louis University School of Medicine.

surgeons founded the Beaumont Hospital Medical College in honor of the city's most distinguished physician, but what plans for a hospital the organizers had in mind is not generally known. A fire destroyed their building, which had previously been occupied by the First Presbyterian Church, in 1889. A new building was erected and equipped within a year, which represented everything modern for that period, and so the school proceeded for more than a decade.

In 1890, another group of progressive and ambitious doctors founded the Marion Sims College of Medicine, the name being taken from the great American gynecologist. This school joined and combined with the last named one in the year 1900 to form the Marion Sims-Beaumont Medical College, which was destined to bear the latter name for only three years. In 1903, the St. Louis University acquired this college by purchase of all its capital stock and since then it has been known as the St. Louis University School of Medicine. The Marion Sims-Beaumont Medical College Building with its adjoining Rebekah Hospital on Grand Boulevard and Caroline Street was continued for teaching purposes but in the remodeling, expansion, and rebuilding of these structures the hospital was replaced by a larger and more modern medical school building. In course of time a long list of private and municipal institutions became available for instruction, but in 1933 the Firmin Desloge Hospital on Grand Boulevard directly opposite the medical school was finished and occupied. It is a teaching hospital in the fullest sense of the term. In 1936, a tunnel was built under Grand Boulevard connecting the two buildings and bringing the laboratories in closer contact with the patients in the hospital.

Thus St. Louis now has two splendid Class A medical schools which are cooperating to the fullest extent with the necessary stimulation to medical progress by a most friendly rivalry.

St. Louis has also three large medical libraries, one in each medical school and the third at the St. Louis Medical Society, 3839 Lindell Boulevard. At this address the "St. Louis Clinics" is also located, where a daily list of surgical operations and other demonstrations in the various hospitals and clinics is published. By inquiring at this office a visiting physician or surgeon can obtain information concerning what is of medical interest in the city, which is truly a medical center.

Book Reviews

Essentials of Dermatology. By Norman Tobias, M.D. Senior Instructor in Dermatology, St. Louis University. 498 pages, illustrated. Philadelphia: J. B. Lippincott Company, 1941. Cloth $4.75.

This is a brief, practical text of modern dermatology. The author has succeeded in presenting the gist of the up-to-date knowledge in this field in a comparatively small volume.

There are 32 chapters which cover the field of dermatology fairly completely. Diseases are discussed as far as possible from the standpoint of internal medicine, and diagnostic features are emphasized. The 151 photographs are well chosen and well reproduced. Treatment is discussed from the standpoint of the general practitioner and for the most part the measures recommended are those which he can carry out.

This is a practical, handy volume of dermatology for the general practitioner and medical student.

Synopsis of Diseases of the Heart and Arteries. By George R. Herrmann, M.S., M.D., Ph.D., F.A.C.P., Professor of Medicine, University of Texas. Second Edition. 468 pages, illustrated. St. Louis: The C. V. Mosby Company, 1941. Cloth $5.00.

The latest diagnostic helps and therapeutic measures are here presented in convenient reference form and in a compact volume. Careful perusal shows the painstaking labor the author has expended in condensing the voluminous output upon cardiology and presenting it in an accessible and attractive form. With the more widespread use of the electrocardiograph the author has included this in the discussion of many heart diseases.

The final chapter on "Military Cardiovascular Examinations and Interpretations" brings the volume up to date for use as reference in the present emergency.

Necropsy. By Bela Halpert, M.D., Assistant Professor of Pathology and Bacteriology, Louisiana State University School of Medicine, and Visiting Pathologist, Charity Hospital of Louisiana at New Orleans. 75 pages. St. Louis: The C. V. Mosby Company, 1941. Cloth $1.50.

This small volume presents a method of postmortem examination based upon the principles of the method used by Ghon in which the organs were removed and examined by systems. The topography and anatomy of the various organs is considered in detail. There is special reference to the regional lymph nodes and tributary blood vessels. The material in the text is arranged in the sequence in which the postmortem examination is performed. The sample postmortem reports appended illustrate the author's method of assembling and evaluating the data.

Infantile Paralysis. A Symposium Delivered at Vanderbilt University, Nashville, Tennessee, April, 1941. 239 pages. New York: The National Foundation for Infantile Paralysis, Inc.

A transcription of a symposium on poliomyelitis given at Vanderbilt University in April of this year, this volume is divided into the various approaches of several authors to the subject. Each gives an exhaustive discussion of the phase in which he has particular knowledge and interest. A chapter on symptomatology and diagnosis would have added to the completeness of the book.

It is recommended to those who desire a comprehensive picture of the research and scientific inquiry into this disease.

The Mask of Sanity. An Attempt to Reinterpret the So-called Psychopathic Personality. By Hervey Cleckley, B.S., B.A. (Oxon.), M.D., Professor of Neuropsychiatry, University of Georgia School of Medicine, Augusta, Georgia. 298 pages. St. Louis: The C. V. Mosby Company, 1941.

This book is a reorientation and a reinterpretation of one of the large wastebasket descriptive diagnoses in psychiatry, the psychopathic personality. It is divided into four main divisions: (1) The Problem; (2) The Material; (3) Cataloguing the Material; and (4) An Attempt at Interpretation. The material is presented in 15 detailed case histories, 9 of which are cases with so-called full clinical manifestations and 6 are case histories of so-called partial clinical manifestations. In

division III current interpretations by various workers are presented; psychopathic personality is compared to other types of personality; and there is a summary of typical and outstanding features of the psychopathic personality. The final discussion is concerned with the questions of (1) What is wrong with these personalities? (2) How does such a change occur? and (3) What can be done?

The author states that the following are typical and outstanding features of the psychopathic personality: he is superficially attractive, with superior general intelligence and with no deterioration throughout the years; he is free from the demonstrable irrationality and commonly accepted symptoms of psychosis and psychoneurosis; he has no sense of responsibility with a total disregard for truth; he is insincere in all activities with no sense of shame; he is unable to learn and profit by experiences, and he has a marked egocentricity with an incapacity for object love; he has a proverty of affect and lacks deep insight into his own problems; he shows little responsiveness to special consideration and kindness; alcoholic indulgence is a prominent feature in his behavior; his social activity usually places him in a disgraceful and ignominious position; his sex life is diminished in affect and is predominantly asexual; he shows a marked inability to follow a life plan and goes out of his way to make a failure of life.

The author believes that the psychopath is fundamentally psychotic with only a mask of sanity. Although there is no latent chaos or personality disintegration he fails completely in the practice of actual life. He demonstrates a persistent lack of ability in becoming aware of the meaning aspect of human life. He does not enjoy the emotional consequences of experience. The author believes that the psychopathic personality can more adequately be called semantic dementia. Dynamically the author believes that the whole life activity of the psychopath is a protracted and elaborate social and spiritual suicide. He believes that the formulation of an active self-destructive tendency can be applied to the psychopath; that the psychopathic personality usually begins early in life, is a developmental phenomenon with a gradual blocking out of all emotional aspects of experience. Therapeutically the author has little to offer. He suggests that special institutions be built for these individuals where they can be studied and intensively investigated.

The problem of the psychopathic personality is ably and logically presented in this book. The case histories are interesting and the style is readable. The formulations and psychodynamics that are discussed will be of value in crystallizing the entity of the psychopathic personality in the minds of all who are interested in abnormal behavior.

Start Today. By C. Ward Crampton, M.D., Major, Medical Reserve Corps, United States Army. 224 pages, illustrated. New York: A. S. Barnes & Co. Cloth $1.75.

Dr. Crampton has had a large experience in writing for the public and now comes forth with a book containing a great deal of wisdom and sage advice. The book is opportune, as it comes at a time when the nation is being made conscious of the necessity for physical fitness and the training of its youth for military service. This book tells the man from the teen age to the three score and ten why he should exercise and diet, and how to do both and want to do it.

Textbook of Pediatrics. By J. P. Crozer Griffith, M.D., Ph.D., Emeritus Professor of Pediatrics in the University of Pennsylvania, and A. Graeme Mitchell, M.D., B. K. Rachford Professor of Pediatrics, College of Medicine, University of Cincinnati. Third Edition. 991 pages, illustrated. Philadelphia: W. B. Saunders Company, 1941.

This work, formerly called "Diseases of Infants and Children," has been completely rewritten and the title changed to "Textbook of Pediatrics."

In its preparation the authors have submitted many of the chapters to specialists for critical comment and suggestions, and have obtained authoritative and up-to-date information.

A chapter on mental growth and development has been added which ably covers this important field. The bibliography included in former editions has been omitted; however, anyone interested can obtain a complete bibliography from the National Research Council.

The book is an invaluable addition to the library of the practitioner as well as a most excellent text for the student.

A Diabetic Manual. By Elliott P. Joslin, M.D., Sc.D., Clinical Professor of Medicine Emeritus, Harvard Medical School, Boston, Massachusetts. 238 pages, illustrated. Philadelphia: Lea & Febiger, 1941. Cloth $2.00.

This manual, written especially for the average diabetic patient, brings up to date the recent advances in the treatment of diabetes mellitus. The use of protamine zinc insulin is well covered.

No patient needs to know so much about his condition as the diabetic. It is essential for him to know how to take care of any emergency that may arise. In order to do this he must be thoroughly familiar with his disease and how to treat it. He must know foods so well that he can make any necessary substitution in his diet on a moment's notice. This manual does much to prepare the diabetic patient to do just these things. It is an indispensable part of the diabetic patient's equipment. Almost every phase of the management of a diabetic is covered, including a discussion of insulin, diets, recipes, insulin reactions, coma, care and use of syringe and needle, composition of various foods and urine tests, and so on.

One chapter is made up of questions and answers. The author has anticipated many of the questions the average patient wants to ask his physician and has answered them in plain language. The book is written in non-technical language which the average patient can understand.

Numerous figures and tables are given. The manual is well indexed.

The Merck Manual of Therapeutics and Materia Medica. Seventh Edition. 1,436 pages. Rahway, New Jersey: Merck & Co., Inc.

This manual contains a great wealth of material for the busy practitioner and enables him to "jog" his memory with very little effort. This edition is similar to those previously issued in make-up, but has been completely revised. New chapters on bile tract disease, obesity, jaundice, circulatory failure and others have been added. A very handy book.

Southern Medical News

ALABAMA

The Alabama State Department of Public Health, in cooperation with the Children's Bureau of the United States Department of Labor, announces Fellowships in Public Health Obstetrics for physicians who have had a minimum of a year's rotating internship and one year of obstetric training. Training provided will embrace field work in already established maternity clinics in rural areas; in the organization of new centers and rotating through the various departments and projects connected with the obstetric aspects of public health. A stipend of $150.00 a month plus travel while in the field is provided. Applications should be sent to Dr. J. N. Baker, State Health Officer, Montgomery.

The Jefferson County Medical Society, Birmingham, held its third annual Postgraduate Seminar on Monday and Tuesday, September 15 and 16. The Seminar drew a large attendance not only from Alabama but from adjoining states. The sessions were presided over by Dr. M. Y. Dabney, Birmingham, President of the Society.

The Alabama State Pediatric Society met in Birmingham, September 17, the program being devoted to papers prepared by Alabama men with the exception of the out-of-territory speaker, Dr. Joseph Stokes, Philadelphia. The Pediatric Society was headed by Dr. M. Vaun Adams, Mobile, President; Dr. Clifford L. Lamar, Birmingham, Vice-President; and Dr. William R. Britton, Montgomery, Secretary-Treasurer. Dr. Lamar was elected President for the coming year.

Dr. William A. Dodson, Jr., Double Springs, has resigned as Health Officer of Winston County to take a position with the U. S. Public Health Service.

Dr. William J. Donald, Brewton, has been appointed an associate in county organization in the State Department of Health, Montgomery, to succeed Dr. W. D. Burkhalter, resigned.

Deaths

Dr. Ben B. Glasgow, Athens, died September 9.

Dr. John Harrison Owens, Duncanville, aged 67, died recently of cerebral hemorrhage.

Dr. Charles A. Poellnitz, Greensboro, aged 65, died September 4.

ARKANSAS

The Tri-State Medical Society (Louisiana, Texas and Arkansas) met in El Dorado, September 23-24, under the presidency of Dr. Rufus B. Robins, Camden. Among the distinguished physicians presenting papers was Dr. M. Pinson Neal, Columbia, Missouri, President-Elect of the Southern Medical Association.

Dr. J. Q. Blackwood, Helena, is taking special public health work at Johns Hopkins University School of Medicine.

Dr. E. E. Estes has been elected President of the Fordyce Country Club.

Dr. L. T. Evans has been elected Treasurer of the Batesville Rotary Club.

Dr. Merl Crow, formerly of Pine Bluff, has moved to Warren where he will be associated with Drs. M. T. and Bruce Crow.

Dr. B. D. Luck, Sr., Pine Bluff, has been elected to fellowship in the International College of Surgeons.

Dr. B. P. Briggs, Little Rock, has been appointed Acting Medical Director of the Crippled Children's Division, State Department of Public Welfare.

Dr. R. V. McCray, Malvern, has been called to active service with the Army Medical Corps and assigned to Fort Leonard Wood, Missouri.

Dr. John W. Dorman, Dyess, has been called to active service as Lieutenant, Medical Corps, U. S. Army, and assigned to Camp Robinson.

Dr. G. P. Slaughter, Cleveland, has been added to the staff of the State Board of Health as Field Consultant in Obstetrics.

Dr. J. L. Bean has been elected Surgeon of the Lincoln Post of the American Legion.

Dr. W. A. Grimmett has been elected surgeon of the Blytheville Post of the American Legion.

Dr. D. A. Dickerson has moved from Gurdon to Caraway.

Dr. Fred H. Krock, Fort Smith, has been promoted to the rank of Lieutenant-Commander, U. S. Naval Medical Reserve Corps.

Dr. W. H. Abington has been elected Surgeon of the Beebe Post of the American Legion.

Dr. Glenn Johnson, Little Rock, has been elected Chief of Staff of the University Hospital.

Dr. Ira Ellis has been elected Surgeon of the Monette Post of the American Legion.

Deaths

Dr. James Edward Bobbitt, Jonesboro, aged 62, died recently of carcinoma of the prostate.

Dr. Sidney Harris, Herbine, aged 74, died recently of cerebral hemorrhage.

Dr. John J. Johnson, Harrison, aged 80, died recently.

Dr. James Henry Strider, Tillar, aged 84, died recently.

DISTRICT OF COLUMBIA

Louis Mackall Medical Society has elected Dr. William B. Marbury, President; Dr. James E. Wissler, Vice-President; and Dr. Darrell C. Crain, Secretary-Treasurer, all of Washington.

Dr. Clifton R. Wallace was appointed Medical Officer in Charge of the National Bureau of Standards, Washington, and began his duties in July.

Dr. Rolla E. Dyer and Dr. Norman H. Topping, U. S. Public Health Service, Washington, have gone to LaPaz, Bolivia, to study the effects of a typhus vaccine developed at the Rocky Mountain Laboratory of the Service at Hamilton, Montana. The test is urgently needed because of the possibility that American troops may have to go into regions of South America where typhus exists and also that the United States may supply Britain with the vaccine.

The National Foundation for Infantile Paralysis announced that the District of Columbia ranked eighth among the ten leading states in the amount of money collected during the President's Birthday Celebration. The national total raised during the 1941 campaign was $2,104,460.

The Medical Advisory Board of the American Committee for Medical Aid to Russia which was organized in Washington by an interested group on August 19 is headed by Dr. Henry E. Sigerist of Johns Hopkins University, Baltimore.

Dr. Walter W. Boyd has been appointed Washington Chairman of Relief Wings, Incorporated, by the Executive Director. This is a national humanitarian service for disaster relief by air.

Dr. Vincent Wilcox, II, Washington, and Miss Mary Frances Bauman, Raleigh, North Carolina, were married recently.

Deaths

Dr. William Tignor Gill, Washington, aged 76, died August 26.

Dr. Kate Breckinridge Bogle Karpeles, Washington, aged 54, died August 15.

Dr. Miles Parker Omohundro, Washington, aged 45, died recently of heart disease.

FLORIDA

Florida East Coast Medical Association will meet in Daytona Beach, December 4 and 5. Dr. J. S. Stewart, Miami, is President, and Dr. J. Ralston Wells, Daytona Beach, Secretary.

Dr. Ralph Gowdy, Miami Beach, has been doing special work at Mayo Clinic, Rochester, Minnesota.

Dr. Maurice J. Rose, Miami Beach, announces the opening of offices in the Mercantile National Bank Building, practice limited to obstetrics and gynecology.

Dr. L. L. Lancaster, Bartow, has been studying at Mayo Clinic, Rochester, Minnesota.

Dr. Frederick LeDrew, Miami, has been appointed a member of the Committee of Psychiatric Standards and Policies of the American Psychiatric Association for a period of five years.

Dr. Courtland D. Whitaker, Marianna, and Miss Mildred Zeagler, Sylvania, Georgia, were married recently.

Dr. Ernest Bostelman and Mrs. Geraldine Olson, both of Fort Myers, were married recently.

Deaths

Dr. Wyatt Barnes, Boca Grande, aged 59, died August 31.

Dr. Nicholas Frederick Feury, Orlando, aged 66, died recently of myocarditis and arteriosclerosis.

Dr. Jacob Alexander Garrard, Bartow, aged 76, died recently.

Dr. Ralph N. Greene, Coral Gables, aged 58, died August 1.

Dr. John Kent Johnston, Tallahassee, aged 53, died recently of heart disease.

Dr. Walter Lewis Scofield, Fort Pierce, aged 58, died recently.

GEORGIA

The Academy of Medicine, the new home of the Fulton County Medical Society, was a dream of the late Dr. Stewart R.

Continued on page 36

Constipation in Infancy

Laxatives

not needed to relieve

Constipation

when the daily feedings are prepared from milk properly modified with

Mellin's Food

Constipation in infancy probably commands the physician's attention more often than any other symptom that points to the need of readjusting a feeding formula.

Constipation is a common complaint and oftentimes is the real reason for a slow gain in weight, restless nights and a fretful, uncomfortable baby.

Infants fed on milk and water in proportions suitable for healthy babies of given age and weight with an amount of Mellin's Food to meet the carbohydrate requirement (*six to eight level tablespoons to the full day's mixture*) are seldom constipated.

Many physicians use Mellin's Food routinely in preparing bottle feedings, for they know from experience that regular stools of good consistency are characteristic of babies fed on milk properly modified with Mellin's Food. These physicians thus avoid much of the trouble associated with infant feeding.

Mellin's Food Co., Boston, Mass.

Samples sent to physicians upon request.

MELLIN'S FOOD: Produced by an infusion of Wheat Flour, Wheat Bran and Malted Barley admixed with Potassium Bicarbonate—consisting essentially of Maltose, Dextrins, Proteins and Mineral Salts.

Continued from page 1092

Roberts of Atlanta, he having been appointed one of the members of the original committee to develop plans for it. So it is the desire of the Society to pay tribute to his leadership and devotion by a lasting memorial within the structure of the building. Any friends wishing to participate in such a tribute should make check payable to the Fulton County Medical Society, marked Building Fund, Stewart R. Roberts Memorial, and mail to Dr. Vernon E. Powell, 768 Juniper Street, Northeast, Atlanta, who is accepting contributions to be used for this purpose.

Plans are being made to establish a memorial for the late Dr. Edward Campbell Davis in the new Academy of Medicine Building, home of the Fulton County Medical Society, Atlanta. Dr. Davis died in 1931. He was Emeritus Professor of Obstetrics and Gynecology at Emory University School of Medicine, a founder of the Davis-Fischer Sanatorium, and in 1928 President of the Fulton County Medical Society.

Announcement has been made by Oglethorpe University, Oglethorpe University, Georgia, of the opening of its medical college. The new College of Medicine is a standard school, fully accredited under the laws and by the authorities of the State of Georgia. Dr. John L. Jacobs, an A.B. of Oglethorpe University, a B.S. of Harvard University, and an M.D. of Harvard Medical College, is Vice-President in charge of the scientific work.

Dr. J. G. Sharpley has been appointed a member of the Committee on Pensions and Charities of the City of Savannah, succeeding the late Dr. William H. Myers.

Dr. Sterling H. Jernigan has located in Sparta and will be associated with his father, Dr. C. S. Jernigan, in the practice of medicine.

Dr. H. Cliff Sauls and Dr. Carter Smith announce the association of Dr. Charles F. Stone, Jr. in the practice of internal medicine with offices in the Medical Arts Building, Atlanta.

Dr. K. C. Walden, Waycross, entertained the members of the Ware County Medical Society at the Atlantic Coast Line Railroad Hospital to dinner recently.

Dr. Bomar A. Olds announces the location of his Atlanta office at 157 Forrest Avenue, Northeast.

Continued on page 38

DOCTOR: *You are invited to attend . . .*

THE OKLAHOMA CITY CLINICAL SOCIETY'S

ELEVENTH ANNUAL FALL CLINICAL CONFERENCE

October 27, 28, 29, 30, 1941

SEVENTEEN DISTINGUISHED GUEST LECTURERS

DR. WALTER C. ALVAREZ, Internal Medicine, Rochester; Professor of Medicine, University of Minnesota (Mayo Foundation).

DR. A. BRUCE GILL, Orthopaedic Surgery, Philadelphia; Professor of Orthopaedic Surgery, University of Pennsylvania.

DR. L. EMMETT HOLT, JR., Pediatrics, Baltimore; Associate Professor of Pediatrics, Johns Hopkins Medical School.

DR. VERNE C. HUNT, Surgery, Los Angeles; Clinical Professor of Surgery, Univ. of Southern California School of Medicine.

DR. HOWARD T. KARSNER, Pathology, Cleveland; Professor of Pathology and Director of Institute of Pathology, Western Reserve University.

DR. FRANCIS E. LeJEUNE, Otolaryngology, New Orleans; Professor of Otolaryngology and Head of Department of Otolaryngology, Tulane University Medical School.

DR. PERRIN H. LONG, Internal Medicine, Baltimore; Professor of Preventive Medicine, Johns Hopkins University Medical School.

DR. JOHN H. MUSSER, Internal Medicine, New Orleans; Professor of Medicine, Tulane University Medical School.

DR. ALTON OCHSNER, General Surgery, New Orleans; Professor of Surgery and Head of Dept. of Surgery, Tulane University Medical School.

DR. EARL D. OSBORNE, Dermatology and Syphilology, Buffalo; Professor of Dermatology and Syphilology, University of Buffalo School of Medicine.

DR. E. D. PLASS, Obstetrics, Iowa City; Professor and Head of Department of Obstetrics and Gynecology, University of Iowa Medical School.

DR. FRED W. RANKIN, President-Elect, American Medical Association; Lexington; Clinical Professor of Surgery, University of Louisville Medical School.

DR. WENDELL G. SCOTT, Radiology, St. Louis; Assistant Professor of Clinical Radiology, Washington University School of Medicine.

DR. ALBERT O. SINGLETON, Surgery, Galveston; Professor of Surgery, University of Texas Medical School.

DR. FRED J. TAUSSIG, Gynecology, St. Louis, Professor of Clinical Obstetrics and Gynecology, Washington University School of Medicine.

DR. GILBERT J. THOMAS, Urology, Minneapolis; Associate Clinical Professor of Urology, Medical and Graduate Schools, University of Minnesota.

DR. HENRY P. WAGENER, Ophthalmology, Rochester; Associate Professor of Ophthalmology, Mayo Foundation, Graduate School of Medicine, University of Minnesota.

General Assemblies Roundtable Luncheons Evening Symposium

Post Graduate Courses Commercial Exhibits

Registration fee of $10.00 includes all the above features.

For further information address Secretary, 512 Medical Arts Building, Oklahoma City

PROVIDE *the Arthritic Patient* *with* RELIEF FROM PAIN . . .

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REG. U.S. PAT. OFF.

A-B-M-C OINTMENT

(ACETYL-BETA-METHYLCHOLINE CHLORIDE)

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A-B-M-C Ointment* relieves arthritic pain because of its local action in increasing the blood supply to the affected part by dilatation of the arterioles and capillaries.

In 88 percent of 96 patients studied, A-B-M-C Ointment provided relief from pain without any untoward effects when used as directed. No urticaria was produced in any case.†

A-B-M-C Ointment is spread, without rubbing, on the affected part and heat is applied for 20 minutes.

Supplied in 1-ounce tubes

†Archives of Physical Therapy, *21*, 12 (Jan.), 1940.

**A-B-M-C Ointment is a trademark of John Wyeth & Brother, Incorporated, for its brand of ointment containing Acetyl-Beta-Methylcholine Chloride 0.25%, menthol, thymol, eucalyptol and methyl salicylate in an emollient base.*

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SAMPLES AND LITERATURE ON REQUEST

John Wyeth & Brother, Inc., Philadelphia

Continued from page 36

Dr. Hal M. Davison, Dr. James C. Thoroughman and Dr. John B. Peschau announce their association for the practice of medicine with offices in the Doctors Building, Atlanta.

Dr. Mason I. Lowance announces the removal of his offices to 215 Doctors Building with practice limited to internal medicine.

Dr. Harold Bernard Levin and Miss Sylvia Leah Meyer, both of Atlanta, were married recently.

Dr. Neuval Virso Cutchins, Jr., Atlanta, and Miss Ann Kaufelt Christian, Richmond, were married recently.

DEATHS

Dr. Linton S. Arthur, Atlanta, aged 57, died recently.

Dr. Luther Benjamin Cook, Coolidge, aged 61, died recently.

Dr. Gaines Richard Thigpen, St. Marys, aged 72, died recently.

Dr. John Arte Usher, Savannah, aged 64, died recently.

Dr. John Griffin Williamson, Rhine, aged 76, died recently.

KENTUCKY

Dr. Fred W. Rankin, Lexington, has been appointed a member of the Medical Advisory Board by the United States Director of Civilian Defense to assist the Medical Division of the Office of Civilian Defense, the purpose of the Board being to keep physicians informed of all medical and public health activities of this organization.

Dr. C. M. Eckler, Williamstown, has been appointed Secretary of the Grant County Medical Society, taking the place of Dr. Lenoir Patrick who resigned to accept the position of Staff Physician in Maternal and Child Health and of Pediatric Consultant to the State Board of Health of Madison, Wisconsin.

Dr. George P. Sprague, Jr., has returned from the East to take over duties as Superintendent and owner of High Oaks Sanatorium, Lexington. For the last ten years he has been the Psychiatrist-in-Charge of the Men's Division of the New York Hospital's Psychiatric Department in White Plains, New York, and previously he was with the Iowa State Psychopathic Hospital and was Assistant Professor of Psychiatry in the Iowa University Medical School.

Dr. Fred C. Dye announces opening of his office in the Brown Building, Louisville, practice limited to anesthesia.

Dr. Robert Cohen, Louisville, and Miss Helen Sylvia Berk, Lafayette, Indiana, were married August 10.

Dr. Robert McClellan James, Louisville, and Miss Lois Livingston, Hazard, were married recently.

DEATHS

Dr. J. L. Allen, Loretto, aged 65, died recently.

Dr. Scott D. Breckinridge, Lexington, aged 59, died August 1.

Dr. A. L. Matthews, Florence, aged 73, died recently of hypertensive cardiovascular renal disease.

Dr. John Alexander Neblett, Louisville, aged 45, died recently of coronary occlusion.

Dr. William Michael Price, Dabney, aged 74, died recently of acute pancreatitis.

Dr. Edwin Franklin Sheppard, Jenkins, aged 48, died recently.

Dr. Livingston Byrons Sheppard, Paintsville, aged 35, died recently.

Dr. Robert B. Scott, Louisville, aged 70, died recently of arteriosclerosis.

Dr. August Schachner, Louisville, aged 75, died recently of hypertensive heart disease.

Dr. Hubert Adolphus Thomas, Princeton, aged 41, died recently of acute endocarditis.

LOUISIANA

The Helis Institute for Medical Research has been created by a trust fund made available by William G. Helis of New Orleans to provide funds for the conduct of medical research and the advancement of the medical sciences. The Institute will establish various clinical and experimental divisions at medical schools and hospitals, and all research carried on will be financed by the Institute. Dr. Carlo J. Tripoli, Assistant Professor of Medicine, Louisiana State University School of Medicine, New Orleans, has been appointed Director of the Institute. The first clinical and experimental division has been established as the Center of Research of Hotel Dieu Hospital, New Orleans.

Continued on page 40

THE NEW YORK POLYCLINIC

MEDICAL SCHOOL AND HOSPITAL

(ORGANIZED 1881)

(The Pioneer Post-Graduate Medical Institution in America)

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General Practitioner

Intensive full time instruction in those subjects which are of particular interest to the physician in general practice. The course covers all branches of Medicine and Surgery.

Proctology, Gastro-Enterology

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IMPORTANT USES FOR KNOX GELATINE IN CONVALESCENCE

Not only is there difficulty in supplying protein to rebuild losses of body protein during illness, but after illness it is important to stimulate appetite by appeal to eye as well as to palate.

During severe illness the loss of body protein may greatly exceed the supply.

- Concentrated protein which is easily digested will help spare body proteins and aid in the repair and replacement of essential tissues.
- The fluid and soft diet during illness and convalescence can easily be supplemented with the Knox Gelatine Drink* (in water, fruit juice or milk), a concentrated source of easily digested protein.
- Knox Gelatine contains a majority of the known amino acids making up proteins, including 7 of the 10 considered "essential." (Amino acid analysis on request.)

*To prepare, pour 1 envelope Knox Gelatine into ¾ glass water, fruit juice or milk, not iced. Let liquid absorb gelatine. Stir. Drink immediately. If gelatine thickens, stir again.

Attractive preparation of food is an important factor in the convalescent diet.

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- With Knox Plain Gelatine the various protective foods can be combined into dishes that are rich in three essential food elements: vitamins, minerals, protein.

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Address.....

Continued from page 38

New Orleans Gynecological and Obstetrical Society and the Louisiana Gynecological and Obstetrical Society will be hosts to the Central Association of Obstetrics and Gynecology at their thirteenth annual meeting which will be held in New Orleans, October 2-4 with headquarters at the Roosevelt Hotel. Dr. H. B. Alsobrook, New Orleans, is Chairman of the local Committee on Entertainment. In addition to the Central Association program, a scientific program on obstetrics and one on gynecology will be presented, Dr. Davil I. Hirsch, Monroe, President of the Louisiana Gynecological and Obstetrical Society presiding over the obstetrical program, and Dr. Lucien A. LeDoux, New Orleans, President of the New Orleans Gynecological and Obstetrical Society, presiding over the gynecological program.

Dr. Charles F. Craig, Colonel, U. S. Army, Retired, San Antonio, Texas, Professor of Tropical Medicine Emeritus, Tulane University of Louisiana School of Medicine, New Orleans, was recently presented by the Scientific Society of San Antonio (Texas) the Franklin Medal for distinguished work in science. Dr. Craig is the first recipient of the medal donated by Colonel and Mrs. W. Lee Hart of San Antonio and named in honor of Mrs. Hart's father, the late Thomas H. Franklin, a fellow of the society.

Dr. Benjamin O. Morrison, formerly of Crowley, has been appointed temporary Director of the Red River Parish Health Unit, filling the vacancy that occurred when Dr. Charles W. Reid, Coushatta, was named Director of the units in Madison and Tensas Parishes with headquarters at Tallulah.

Dr. George M. Leiby, formerly Director of Preventive Work against Venereal Diseases in the District of Columbia and in North Carolina, has been appointed Director of the Division of Preventive Medicine in the Louisiana State Board of Health.

Dr. Edgar Sevier Lotspeich, Jr., New Orleans, and Miss Jane Carrington, Richmond, Virginia, were married recently.

Deaths

Dr. James C. Denman, Franklinton, aged 66, was killed September 13 in an automobile collision.

Dr. Joseph James Wymer, Waveland, aged 62, died August 9.

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Guest Lecturers: Drs. Wm. L. Benedict, Edwin B. Dunphy, Harry S. Gradle, Walter I. Lillie, S. Hanford McKee, Avery DeH. Prangen, Algernon B. Reese, Meyer Wiener.

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AVIATION MEDICINE AND AVIATION OPHTHALMOLOGY

FEBRUARY 5th-7th, 1942, Inclusive

Guest Lecturers: Lt. Col. David N. W. Grant, M.C., U.S.A.; Lt. Col. Frederic H. Thorne, M.C., U.S.A.; Major John M. Hargreaves, M.C., U.S.A.; Major M. S. White, M.C., U.S.A.; Capt. J. C. Adams, M.C., U.S.N.; Capt. J. R. Poppen, M.C., U.S.N.; Comdr. Eric Liljencrantz, M.C., U.S.N.; Lt. Comdr. Leon Carson, M.C., U.S.N.; Dr. Hodges McKnight, American Air Lines; Col. A. D. Tuttle, M.D., United Air Lines; Dr. W. Randolph Lovelace II, Mayo Clinic; Dr. Ross A. McFarland, Harvard University. Fee $35.00

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MARYLAND

American Association of the History of Medicine has established the William Osler Medal to be awarded annually to the author of the best student essay submitted to the Association and it will be awarded for the first time at the eighteenth annual meeting to be held in Atlantic City, May 3-5, 1942. The medal is being awarded to stimulate interest and research in medical history among students of the universities of the United States and Canada. Essays are to be sent before November 1, 1941 to the Secretary of the Association, Dr. Henry E. Sigerist, Institute of the History of Medicine, 1900 East Monument Street, Baltimore.

Dr. Huntington Williams, Health Commissioner of Baltimore, went to England in July to study disaster precaution and technics of medical care after bombing. Dr. Williams was one of a group representing the Office of Civilian Defense.

Dr. William L. Quennel, Superintendent of the Highland Park General Hospital, Highland Park, Michigan, has been appointed Director of the Union Memorial Hospital, Baltimore, succeeding the late Dr. Clyde D. Frost.

Deaths

Dr. Clyde DeWitt Frost, Baltimore, aged 49, died recently.

Dr. Norman Thomas McLean, Pearson, aged 65, died recently.

Dr. Hiram Leslie Reckard, Baltimore, aged 69, died recently of uremia.

MISSISSIPPI

Dr. L. L. Lumsden, Nashville, Tennessee, who has had wide experience in the epidemiology of poliomyelitis, has been assigned to Mississippi by the National Foundation for Infantile Paralysis. While in the State he is to be associated with the Division of Preventable Disease Control of the State Board of Health at Jackson.

Dr. Raymond L. Mayer, formerly Health Officer of Sunflower County, has been appointed Director of the Claiborne County Health Department.

Dr. James F. Bradley has been appointed Health Officer of Amite County, succeeding Dr. J. A. Sproles who has been appointed Director of the Marion County unit. Dr. Sproles succeeds Dr. J. M. Campbell who has been appointed Director of the Tallahatchee County Health Department.

Dr. C. R. Gillespie, formerly Director of Sunflower County Health Unit, is now connected with the Field Advisory Unit of the State Board of Health.

Deaths

Dr. Jacob Moon Anderson, Sardis, aged 71, died July 8.

Dr. Wiley Sharp Cranford, Jr., Ellisville, aged 58, died recently of chronic osteomyelitis.

Dr. J. P. Corn, Monticello, died August 6 of a heart attack.

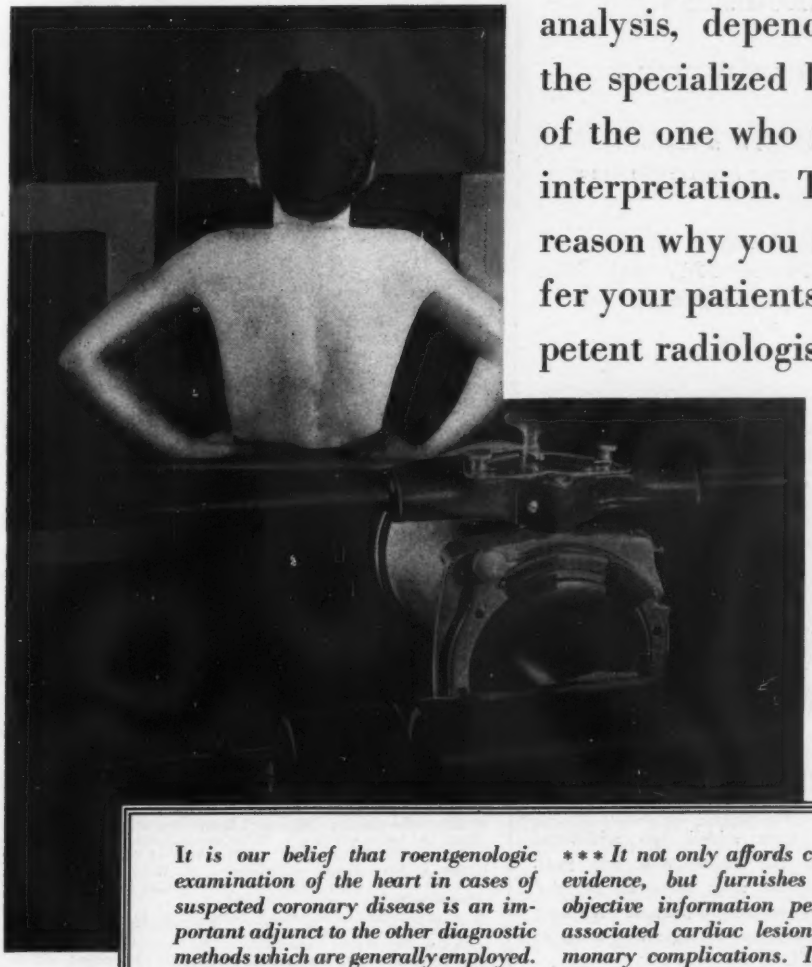
Dr. W. C. Norris, Quitman, aged 59, died September 3.

MISSOURI

Kansas City Southwest Clinical Society will hold its nineteenth annual fall clinical conference in Kansas City, October 6-9.

Continued on page 42

The diagnostic value of any radiograph is, in the final analysis, dependent upon the specialized knowledge of the one who makes the interpretation. This is the reason why you should refer your patients to a competent radiologist.



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R. B. H. GRADWOHL, M.D.
Director

Continued from page 40

Second Congress on Obstetrics and Gynecology will be held in St. Louis, April 6-10, 1942.

Kansas City Society of Ophthalmology and Otolaryngology at a recent meeting presented a gold medal to Dr. John L. Myers, Kansas City, this in appreciation of his work in the Society.

Dr. Harry L. Alexander, Associate Professor of Clinical Medicine at Washington University School of Medicine, St. Louis, has been appointed Professor of Clinical Medicine and acting head of the Department of Internal Medicine, succeeding Dr. David P. Barr who resigned to accept an appointment at Cornell University Medical College, New York City.

Dr. James A. Atkins, Lamar, was elected a District Governor of Rotary International at a meeting of the organization in Denver. Dr. Atkins' district includes Missouri, Kansas, Arkansas and Oklahoma.

Dr. H. R. DuPuy, formerly of Oakland, Maryland, is the new Health Officer of Berkeley County.

Dr. T. H. Millman, Pineville, has been appointed Director of Lovan County's venereal disease project.

Dr. W. A. Bevacqua, Health Officer of Wood County, resigned September 1 to enter private practice in Parkersburg.

DEATHS

Dr. Wheeler Davis, Kennett, aged 72, died recently.

Dr. Emmett F. Cook, St. Joseph, aged 68, died recently of heart disease and cerebral hemorrhage.

Dr. Max Aaron Goldstein, St. Louis, aged 71, died recently of cerebral hemorrhage.

Dr. John Whitworth Harrison, Kansas City, aged 64, died recently of chronic myocarditis.

Dr. William Spencer Love, Charleston, aged 58, died recently of a malignancy of the left lung.

Dr. William Franklin Pitt, Hayti, aged 49, was killed recently in an auto accident.

Dr. George Vaughan Poynor, South West City, aged 85, died recently of carcinoma of the mouth and throat.

Dr. Warren Robert Rainey, St. Louis, aged 54, died recently of Rocky Mountain spotted fever.

Dr. Squire H. Redmon, Tipton, aged 87, died recently of chronic interstitial nephritis.

NORTH CAROLINA

Southern Tuberculosis Conference held its annual meeting at the George Vanderbilt Hotel, Asheville, September 15-17 under the presidency of Dr. H. Frank Carman, Dallas, Texas.

Duke University School of Medicine and Duke Hospital will have a Symposium on Problems of Civil and Military Emergencies in the Page Auditorium on the West Duke Campus, October 16-18, the program being presented by nationally known authorities. On Friday evening, October 17, there will be a buffet dinner in order that doctors attending the Symposium may meet the speakers and have a social evening together. Dr. Lenox D. Baker is chairman of the Symposium Committee.

American College of Physicians will hold its annual regional meeting in Chapel Hill, October 31-November 1.

Buncombe County Medical Library has recently moved to larger and better quarters in the Arcade Building, Asheville.

The Office of the North Carolina Medical Journal is now at Bowman Gray School of Medicine of Wake Forest College, Winston-Salem. Manuscripts and communications should be addressed there.

A city blood plasma bank is being set up in Asheville by the Buncombe County Medical Society, the five Asheville hosoitals, and the City Health Department. The hospitals will supply the necessary laboratory equipment, the Health Department will do the laboratory work, and a committee from the Medical Society will be in charge of the project. Dr. G. Farrar Parker, Asheville, is head of the committee.

Dr. A. T. Miller, Instructor in Physiology, University of North Carolina, Chapel Hill, taught in the Department of Physiology at the University of Michigan School of Medicine for the summer session.

Dr. Richard Query, Charlotte, has been certified by the American Board of Internal Medicine.

Dr. R. B. McKnight, Charlotte, has been certified by the American Board of Surgery.

Dr. George R. Benton, Jr., formerly of Goldsboro, is at Station Hospital, Camp Livingston, Louisiana.

Dr. Parker C. Hardin has moved from Monroe to the Stark General Hospital, Charleston, South Carolina.

Continued on page 44

A TEXT BOOK OF MODERN X-RAY TECHNIC AND PROCEDURE

Containing more than 200 pages of comprehensive information on every phase of radiographic technic, the new edition, "Outline of Modern X-Ray Technic"—will be welcomed by everyone engaged in x-ray procedure.

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Continued from page 42

Dr. R. W. Prichard has moved from Kinston to Pink Hill.

Dr. Paul M. Deaton has become a member of the medical staff of the H. F. Long Hospital, Statesville, having just finished an internship in the Lankenau Hospital in Philadelphia.

Dr. Walter J. Lackey, Fallston, has been taking an intensive course in rectal diseases in Chicago.

Dr. Oscar Lee Miller, Charlotte, has been elected to honorary membership in the Argentine Society of Surgeons.

Dr. Oscar Billeter, formerly of Chicago, is the new Resident Surgeon at Hugh Chatham Hospital, Elkin.

Dr. William St. Julien Jervey and Miss Susan Amelia Sprague, both of Tryon, were married recently.

Dr. William Henry Bandy, Lincolnton, and Miss Pauline Pope, Linwood, were married recently.

Dr. Claude Ernest Simons, Wilson, and Miss Margaret Smith Moye, Goldsboro, were married August 5.

Dr. Thomas J. Holt, Jr., Warrenton, and Miss Lela Manning, Bainbridge, Georgia, were married recently.

Dr. George Ashby Winstead and Miss Jacqueline Gainer Denson, both of Rocky Mount, were married recently.

Deaths

Dr. Joseph M. Hodges, Boone, aged 75, died recently of myocarditis.

Dr. William Bernard Kinlaw, Rocky Mount, aged 45, was killed recently in an automobile accident.

Dr. George H. Ross, Durham, aged 77, died recently of bronchopneumonia and hypertensive cardiovascular disease.

OKLAHOMA

Oklahoma City Clinical Society will hold its eleventh annual fall clinical conference October 27-30.

The following members of the Oklahoma State Medical Association have been appointed by President Finis W. Ewing to membership on a Committee to study prepaid surgical benefits: Dr. John F. Burton, Oklahoma City, Chairman; Dr. V. C. Tisdale, Elk City; Dr. C. W. Arrendell, Ponca City; Dr. Benjamin A. Davis, Cushing; and Dr. Gifford H. Henry, Tulsa.

An Advisory Committee has been appointed by the Oklahoma State Medical Association to act in an advisory capacity to the State Department of Public Welfare and members are: Dr. Charles R. Rountree, Chairman, Oklahoma City; Dr. Alfred R. Sugg, Ada; Dr. Robert M. Shepard, Tulsa; Dr. Frank Clinton Gallaher, Shawnee, and Dr. Frederick Redding Hood, Oklahoma City.

Dr. Ray H. Ballard, for the past year Director of the Cleveland County Health Department, Norman, on August 1 took over his new duties as Student Health Director at the University of Wyoming, Laramie, Wyoming.

Dr. Paul Kernek, formerly of Oklahoma City, is practicing medicine with Dr. Victor W. Pryor in the Pryor-Johnston-Kernek Hospital-Clinic in Holdenville.

Dr. Charles E. Leonard, formerly of Fort Supply and Chicago, Illinois, has joined the staff of the Coyne-Campbell Clinic, Oklahoma City, to carry on work in psychiatry and neurology.

Dr. John K. Coker, formerly of Duncan, has moved to Clovis, New Mexico, where he will carry on a general practice and also be associated with the Clovis Memorial Hospital.

Dr. Paul W. Smith has been appointed to fill the vacancy of Dr. Benedict E. Abreu, Instructor in Pharmacology, University of Oklahoma School of Medicine, who resigned to accept the appointment of Assistant Professor of Pharmacology at the University of Georgia School of Medicine. For the past three years Dr. Smith was Assistant Professor of Physiology at the University School of Medicine, Memphis.

Dr. Lowell L. Stokes, Anadarko, Health Officer of the Caddo County Health Department, has been placed in charge of a newly created unit in Okmulgee County. Dr. Hugh H. Hawley, Jr., Anadarko, is now in charge of Caddo County.

Dr. William A. Loy, Director of the Department at Ardmore and for the past year doing graduate work in public health at Harvard University, Boston, has been assigned to Cleveland County.

Dr. Ferdinand R. Hassler, Jr., Shawnee, who recently completed a course of study at Johns Hopkins School of Hygiene and Public Health, Baltimore, Maryland, has been appointed Director of Laboratories for the Oklahoma State Department of Health,

Continued on page 46

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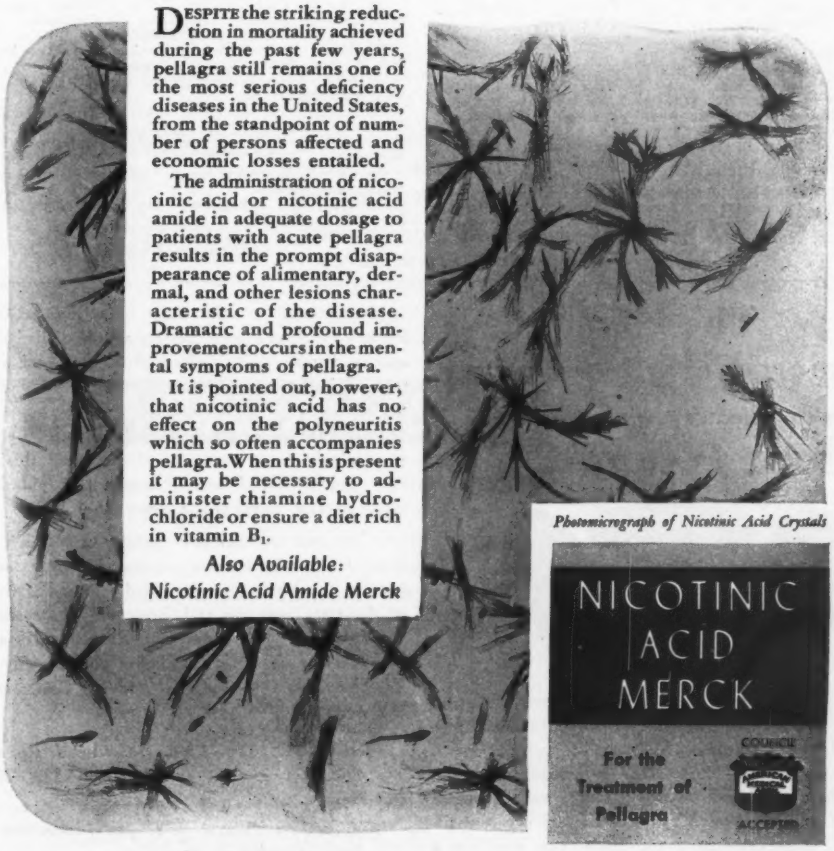

Photomicrograph of Nicotinic Acid Crystals

Literature on Request

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Continued from page 44

Oklahoma City. He was formerly Director of the city-county health department at Shawnee.

Dr. Tilden Hendricks Phipps, Jr., Hollis, and Miss Anna Dorothea Righs, Tampa, Florida, were married August 9.

Deaths

Dr. John W. Lee, Tulsa, aged 68, died recently.

Dr. William Blaine Clark, Tulsa, died recently of nephritis, hypertension, heart disease and arteriosclerosis.

Dr. Charles R. Silverthorne, Woodward, aged 70, died recently of cerebral hemorrhage.

Dr. Ernest Sullivan, Oklahoma City, aged 59, died recently of heart disease.

Dr. Robert Leland Westover, Okmulgee, aged 68, died recently of myocarditis.

SOUTH CAROLINA

The seventh annual Piedmont Post Graduate Clinical Assembly was held at the Anderson County Hospital, Anderson, September 9-11, under the presidency of Dr. Joseph Decherd Guess of Greenville.

Second District Medical Society has elected Dr. E. W. Tucker, Johnston, President; Dr. W. A. Hart, Columbia, Vice-President; and Dr. William Weston, Jr., Columbia, Secretary.

Dr. J. I. Converse, a recent resident at the Greenville General Hospital, and Dr. J. W. Bell, a recent interne, have located in Greenville.

Dr. V. M. Rhinehart has opened an office for general practice of medicine at Newberry.

Dr. Malcolm Mosteller has opened an office in Columbia for practice of radiology.

Dr. E. M. Rice, Director of the Health and Sanitation Division of the South Carolina Public Service (Santee-Cooper) has gone to Washington for service in the Army and will hold rank of Major and Director of an Army school.

Dr. T. B. Reeves, Greenville, and Miss Julia Smyth, Hendersonville, North Carolina, were married recently.

Dr. Frank P. Gaston and Miss Dora Jones Dunlap, both of Rock Hill, were married recently.

Dr. Norman Douglas Ellis, Jr., Martin, and Miss Martha Alice Bennett, Dillon, were married recently.

Dr. Harold Rawling Pratt-Thomas and Miss Mary Porcher Douglas, both of Charleston, were married July 2.

TENNESSEE

Dr. John M. Lee, Nashville, has been appointed State Health Director to organize and direct the National Youth Health Program, a nationwide health project of the National Youth Administration for youths aged 17 to 25 years who are enrolled in the out-of-school work program now being organized in Tennessee. The Advisory Committee appointed is composed of Dr. H. H. Shoulders, Chairman; Dr. Charles M. Hamilton, Dr. Leonard W. Edwards, Dr. Wilson Carter Williams and Dr. Lee, all of Nashville.

Dr. James A. Price, Oakville, has resigned as Medical Director of the Oakville Memorial Sanatorium. He has been in charge of the city and county institution since it opened in 1921. Dr. Price is succeeded by Dr. Felix A. Hughes, Jr., Nashville.

Dr. Julian E. Williams, Kingsport, has been appointed Health Officer of Sullivan County during the absence of Dr. J. W. Erwin, Blountville, in military service.

Dr. William O. Baird, Henderson, has been appointed Commissioner of State Institutions by the Governor.

Dr. Howard King and Dr. C. M. Hamilton, Nashville, announce the association of Dr. Robert N. Buchanan, Jr., practice limited to dermatology and syphilology.

Dr. Joseph Drake Anderson, Franklin, and Miss Anne Elizabeth Woodard, Spring Hill, were married recently.

Dr. Sol L. Lowenstein and Miss Corinne Cohn, both of Nashville, were married July 2.

Dr. L. A. Wilcox has moved from Memphis to Turner Clinic, Piggott, Arkansas.

Dr. R. D. Henderson has moved from Memphis to Olive Branch, Mississippi.

Dr. Chas. J. Deere has moved from Memphis to Fort McPherson, Atlanta, Georgia.

Dr. Harry W. Hollingsworth has moved from Jonesboro to Leona Mines, Virginia.

Continued on page 48

The New Emmert-Gellhorn Pessary

An improved device for treatment of inoperable uterine prolapse

For use in cases of inoperable uterine prolapse, this new pessary is offered as an improvement upon the well-known Gellhorn Pessary, long used with great success. The Emmert-Gellhorn Pessary is made of one solid piece of Neicomold, a synthetic material that may be boiled. The material is unbreakable and stays smooth in use since it is unaffected by the genital secretions. Neither does it affect or irritate the vaginal mucosa. The shape is approximately that of a mushroom, with the upper surface slightly depressed.

Instead of a solid stem, the Emmert-Gellhorn Pessary employs a stem having a hole drilled throughout its length. This offers the considerable advantage of drainage, preventing accumulation of dammed-up secretions. This permits fewer removals of the pessary for cleaning, of great benefit to the patient, particularly since many of those using this pessary are aged and find such frequent manipulation and visits to the physician a severe handicap.

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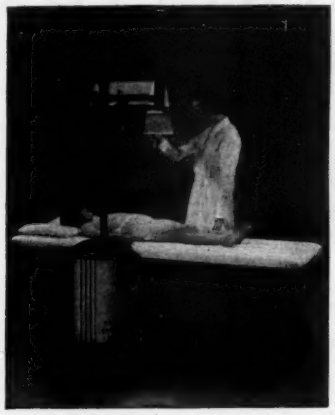

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Continued from page 46

Dr. S. S. Brown has moved from Jellico to Pleasant View, Kentucky.

Dr. A. T. Hicks has moved from Bruceton to Camden.

Dr. W. D. Anderson has moved from Memphis to Tuscaloosa, Alabama.

Dr. S. L. Stephenson, Jr., of Savannah, is now at Sation Hospital, Fort Jackson, South Carolina.

Dr. R. B. Chrisman of Memphis is now at Station Hospital, Camp Forrest.

Dr. H. H. Hudson has moved from Cleveland to Memphis.

Dr. O. H. Clements has moved from Palmer to Bethesda Hospital, Cincinnati, Ohio.

Dr. W. W. Winters of Nashville is now at Western State Hospital, Bolivar.

Dr. C. E. McCarthy of Murfreesboro is now located at the City Hospital, Columbus, Georgia.

Dr. Frank C. O'Connor has moved from Chattanooga to Rossville, Georgia.

Dr. I. E. Philips, Tazewell, has moved to Worley, Kentucky.

Dr. Frank F. Ellis, Jr., of Old Hickory is now in the Seventy-Ninth Field Artillery at Fort Bragg, North Carolina.

Deaths

Dr. Eustace Cosmo Mason, Quebeck, aged 72, died recently following an operation for appendicitis.

Dr. Frank J. Runyon, Clarksville, aged 79, died August 6.

Dr. Herman Reynolds, McMinnville, aged 63, died recently of coronary thrombosis.

Dr. B. Whit Sutton, Huntland, aged 75, died recently.

Dr. Charles Anthony Walters, Santa Fe, aged 70, died recently of myocarditis.

TEXAS

Texas Ophthalmological and Otolaryngological Society will hold its next meeting at San Antonio, December 12-13.

Texas Surgical Society will hold its next meeting at Galveston, October 6 and 7.

Continued on page 50

Classified Advertisements

RATES for insertion in the Classified Column are as follows: $2.00 minimum, which includes the first 50 words; for each word in addition to the original 50 words, the charge is 3c.

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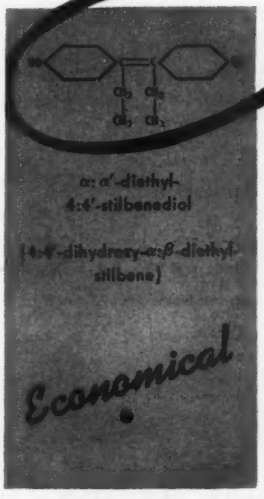

CRYSTALLINE SYNTHETIC ESTROGEN

The new synthetic estrogen, Stilbestrol, now made generally available for the first time, has proved effective when given by mouth as well as by injection. A long awaited advancement is thus realized in an important field of hormone therapy. Moreover, Stilbestrol is much more economical to use than synthetic natural estrogens or extracts from animal sources.

INDICATIONS: Stilbestrol is indicated wherever an estrogenic effect is desired. It has been found of particular value in relieving the symptoms of the menopause, in senile vaginitis, and in gonorrheal vaginitis in children. It will relieve or prevent painful engorgement of the breasts during suppression of lactation.

HOW SUPPLIED

Tablets, 0.1 mg., 0.5 mg. and 1 mg., in bottles of 50, 250 and 1000.

Suppositories, 0.1 mg. and 0.5 mg., in boxes of 5 suppositories.

Ampules (Stilbestrol in Oil), 0.5 mg. in 1 cc., and 1 mg. in 1 cc., each in boxes of 5 and 25 ampules.

Doctor: Watch your mail for Winthrop's booklet giving the essential details regarding this new therapeutic agent: Chemical and pharmacologic data, review of clinical reports, discussion of contraindications, side effects, methods of administration and dosage table.

*The Council on Pharmacy and Chemistry of the American Medical Association has recently adopted the name "Diethylstilbestrol" as the common nonproprietary designation.

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Continued from page 48

Texas Public Health Association will hold its next meeting at Corpus Christi, November 3-5.

Southwest Texas District Medical Society has elected Dr. W. E. Whigham, McAllen, President; Dr. C. M. Kent, Kennedy, Vice-President for Fifth District; Dr. H. M. Austin, Laredo, Vice-President for Sixth District; and Dr. Charles William, Laredo, Secretary-Treasurer.

Jefferson Davis Hospital Intern Alumni has elected Dr. L. M. Shipp, Henderson, President; Dr. Herbert Duke, Goose Creek, Vice-President; and Dr. John M. Filippone, Houston, Secretary-Treasurer.

Physicians of Beaumont recently voted unanimously for a proposed charity hospital for that city.

Dr. C. A. Wyatt and Dr. L. Redding are constructing a new clinic building at Marshall at a cost of approximately $8,000.

Haskell Clinic Building, built by Dr. Earnest M. Kimbrough and Dr. Gordon Phillips at a cost of approximately $14,000, was opened recently.

A new hospital-clinic building at Freeport is being built by Dr. George D. Reeves, Dr. W. T. Galloway and Dr. A. O. McCary at a cost of $30,000.

U. S. Naval Air Station Hospital, Corpus Christi, was recently dedicated and Dr. N. D. Buie, Marlin, President of the State Medical Association, gave the principal address.

A new clinic building is being built at Tyler by Dr. H. R. Coates and Dr. M. V. Gafney at a cost of approximately $30,000.

Dr. William Carlton Farmer, San Antonio, was recently made an honorary member of the National Tuberculosis Association.

Dr. John H. Finn, Refugio, was recently appointed Health Officer of Refugio County, succeeding Dr. H. B. Woods, who has been called to Army service.

Dr. C. H. Mason, El Paso, was recently appointed a member of the El Paso City-County Board, filling a vacancy caused by the resignation of Dr. T. A. Black.

Dr. Charles F. Craig, Colonel, Medical Corps, U. S. Army (Retired) San Antonio, was recently awarded the Franklin Medal for distinguished work in science by the Scientific Society of San Antonio. He is the first recipient of the medal donated by Col. and Mrs. W. Lee Hart of San Antonio and named in honor of Mrs. Hart's father, the late Thomas H. Franklin.

Dr. Henry C. Wilson, Greenville, was recently named the Director of Tyler-Smith County Health Unit, replacing Dr. R. L. Cherry, who has been appointed Field Director for the county health units for the State Department of Health.

Dr. B. A. Wight, Kermit, was recently appointed Health Officer of Gregg County.

Dr. E. H. Lyle, Wink, is taking postgraduate work in Rochester.

Dr. Elmer Haynes, Rusk, has been taking postgraduate work at the University of Wisconsin.

Dr. S. S. Munger, Marlin, was recently elected Health Officer of that city.

Dr. A. L. Nelson, Nacogdoches, has recently returned from postgraduate study in New York and other clinical centers in the East.

Dr. E. P. Shelton, Dripping Springs, was recently honored at a picnic attended by six hundred friends, celebrating his fiftieth anniversary in the practice of medicine. A purse of $100 was presented to Dr. and Mrs. Shelton.

Dr. Walter R. McBee, Executive Director of Group Hospital Service of Oklahoma, has been appointed to a similar position with the state group in Texas.

Dr. Harold C. Shilling, Fort Worth, has been made Director of the Hunt County Unit.

Dr. C. J. Donaghey, Trenton, has returned from postgraduate study in Los Angeles.

Dr. Herbert Locke, Gainesville, has been taking postgraduate study work in Los Angeles.

Dr. W. L. Souther, Waco, was elected State Department Surgeon, Veterans of Foreign Wars, at the recent state meeting of that organization in Fort Worth.

Dr. Minnie L. Maffett, Dallas, was reelected President of the National Federation of Business and Professional Women's Clubs at the bi-annual convention of that organization held recently in Los Angeles.

Dr. Hal White Hardin, Dallas, and Miss Ellen Hughes Merscher, Wilmington, Delaware, were married recently.

Dr. Jack Clark McCurdy and Miss Isabelle Davidson, both of Marshall, were married recently.

Dr. Donald J. Barnett has moved from Marshall to Fort Worth.

Continued on page 52

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Activation of the Entire Intestinal Tube

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Normalization of Evacuation

A Dual Acting Physiologic Corrective

Correction of chronic constipation calls for activation of the entire tract rather than irritation of the large bowel alone.

Cholmodin—deoxycholic acid and extract of aloes—provides this needed dual action. Deoxycholic acid, a bile derivative, promotes increased peristalsis in the small bowel; at the same time it activates the extract of aloes, reducing the period of latency, and permitting a smaller dosage to be used.

One or two Cholmodin tablets at night produce easy evacuation of formed feces. Administration over an appropriate period of time usually results in spontaneous normal defecation—true physiologic correction.

Clinical test samples on request. Prescribe boxes of 50 tablets.

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WAS AND STILL IS THE OUTSTANDING SOURCE OF THE FULL VITAMIN B COMPLEX SUPPLYING THE FULL OF GOLDBERGER'S P-P (PELLAGRA-PREVENTIVE) FACTOR

Since the Mississippi flood in 1928, Vegex-Vitafood Dried Brewers' Yeast has increasingly become the outstanding source of Goldberger's P-P (pellagra-preventive) factor, particularly among health departments and physicians.

Initially, the President of the Company, with a long experience in food and drug administration, the author of one of the first, if not the first, of the laws against medical advertising frauds, determined to and did place the use of this yeast entirely under the direction of physicians and health departments. It has been, with the physician's other treatment, a dependable aid in lowering the pellagra death rate as comparative vital statistics are showing.

And Then

Another outstanding thing has been done. It has been demonstrated that when a product is supplied at a price which people of low incomes can pay, supplied through the wholesaler and retailer and then all treatment directed to the neighborhood physician and health department, a fatal scourge, even during the depression, through the private practitioner, is being conquered.

The Two Types

There are two types of Vegex-Vitafood Dried Brewers' Yeast, the Red Label has the hop flavor removed; the Green Label retains the hop flavor. Both types are packed in one-half pound, one, two and five pound cans or fifty and one hundred pound bags.

This yeast assays 50 International B^1 units per gram and 40 Sherman-Bourquin B^2G (riboflavin) units per gram and supplies the whole of the vitamin B complex. It is high in Goldberger's P-P (pellagra-preventive) factor including the nicotinic acid fraction.

Suggested Administration

A simple and satisfactory way for administration is a tablespoonful stirred into a glass of cold water. The debittered type is furnished in 6 grain tablet form. However, in pellagra, the powder is preferable since the daily dose would require from thirty to sixty tablets.

Vegex

Vegex is the condensed extract of approximately the whole contents of the yeast cell in a more palatable and more available form. It is meat-like in flavor—but meat free—giving wide and tasty food uses. The simplest way of serving Vegex is to dissolve a scant teaspoonful of the paste into a cup of hot water—chill in summer—or hot milk.

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Samples for clinical or professional use will be sent on request.

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Continued from page 50

Dr. John H. Barrett has moved from Palestine to New York City.

Dr. R. W. Beck has moved from Galveston to Madison, Wisconsin.

Dr. E. R. Crews has moved from Austin to Lubbock.

Dr. J. E. Dailey has moved from Houston to Naval Air Station, Corpus Christi.

Dr. Sam H. Dryden has moved from Fort Worth to Fort Sam Houston.

Dr. O. J. Emory has moved from Rule to Fort Worth.

Dr. Stephen Fairbanks has moved from Gainesville to Luther, Michigan.

Dr. T. Alvin Fears has moved from Beaumont to Naval Air Station, Corpus Christi.

STAIRWAY ELEVATOR

By use of the LEVERATOR, a hand elevator, a person with a weak heart may be assisted in going up a stairway one or two steps at a time without the strain of climbing.

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DAILY LOG

Dr. R. E. Fisher has moved from Dallas to Charleston, South Carolina.

Dr. J. O. Gooch has moved from Shamrock to Fort Sam Houston.

Lt. Lawrence L. Griffin has moved from Galveston to New Orleans, Louisiana.

Dr. J. J. Hanna has moved from Quanah to Glen Rose.

Dr. W. Ray Hanna has moved from Gladewater to Sulphur Springs.

Dr. J. D. Hartzo has moved from Atlanta to McLeod.

Dr. Elmer Haynes has moved from Rusk to Madison, Wisconsin.

Dr. Harry Jacobson has moved from Post to Ellington Field, Houston.

Dr. Jack Kinell has moved from Corpus Christi to Houston.

Dr. James W. Long has moved from Port Arthur to Camp Wallace, Hitchcock.

Dr. Paul R. Maxwell has moved from Harlingen to Mansfield, Ohio.

Dr. R. L. McLaughlin has moved from Fort Worth to Dallas.

Dr. Charles R. Potts has moved from San Angelo to Houston.

Dr. W. H. Williams has moved from Abilene to San Antonio.

Dr. J. E. Willerson has moved from Austin to San Antonio.

Deaths

Dr. Raleigh William Baird, Dallas, aged 71, died recently.

Dr. Max Benjamin Brandenberger, Seguin, aged 59, died recently of heart disease.

Dr. John Edward Cannon, Celeste, aged 83, died recently of arteriosclerosis.

Dr. Claude Levi Edge, Crosbyton, aged 53, died recently.

Dr. John W. Duncan, Jourdanton, aged 67, died recently of cerebral hemorrhage.

Dr. Jacob Moore Frazier, Belton, aged 84, died recently.

Dr. Dallas Curtice Johnson, San Antonio, aged 43, died recently of pulmonary tuberculosis.

Dr. Atchilous Bramwell Moore, Greenville, aged 87, died recently.

Dr. Antonio Garcia Padilla, Brownsville, aged 71, died recently.

Dr. Bernard Rubenstein, Dallas, aged 51, died recently of coronary occlusion.

Dr. Eugene William Rufus Williams, Celeste, aged 74, died recently of carcinoma of the liver.

VIRGINIA

Augusta County Medical Association has elected Dr. H. B. Webb, Waynesboro, President; Dr. James B. Pettis, Staunton; Dr. H. J. Morton, Stuarts Draft, and Dr. L. C. Brown, Staunton, Vice-Presidents; Dr. J. E. Womack, Staunton, Treasurer; and Dr. J. H. Thomas, Greenville, Secretary.

Halifax County Medical Society has elected Dr. L. P. Bailey, Nathalie, President; and Dr. Wm. C. Brann, South Boston, Secretary-Treasurer, reelected.

Dr. Francis N. Taylor, Petersburg, has been appointed Chairman of the Health and Welfare Committee and also a member of the Program Committee of the Petersburg Lions Club for the coming year.

Dr. Meade C. Edmunds, Petersburg, has been named a member of the Sight Conservation Committee.

Dr. James McCaw Tompkins, Richmond, has been appointed by

Continued on page 54

LaMOTTE BLOOD CHEMISTRY SERVICE

New LaMotte Outfit for the Determination of Sulfanilamide, Sulfapyridine, Sulfathiazole, Sulfaguanidine and Sulfadiazine in Blood and Urine

Latest improved procedure employs N (1-Naphthyl) ethylenediamine dihydrochloride and Ammonium Sulfamate in the coupling reaction. Determines free and combined form in blood and urine. This simple procedure enables one to determine one or all five of these drugs in a minimum amount of time. Outfit is complete with instructions. Price $23.50 F. O. B. Towson, Baltimore, Md.

This Service includes a series of similar outfits for conducting the following accurate tests: Blood Sugar, Blood Urea, Icterus Index, Phenolsulphonphthalein Urine pH, Blood pH, Gastric Acidity, Calcium-Phosphorus, Blood Bromides, Blood Proteins, Urinalysis.

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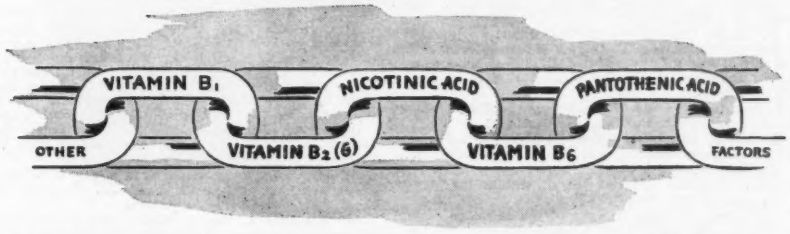

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Continued from page 52

the Governor as a member of the Board of Visitors of the Medical College of Virginia, succeeding the late Dr. W. H. Ellerson, the appointment being for life.

Dr. T. J. Hughes, Roanoke, attended a convocation of the International College of Surgeons in Mexico City recently at which time the Degree of membership was conferred upon him.

Dr. Q. H. Barney was recently elected Commander of the American Legion Post 134 and Dr. Carleton Moorman First Vice-Commander, both of Altavista.

Dr. Marion K. Humphries, Jr., Farmville, who completed his residency in ophthalmology and otolarynogology at the University of Virginia Hospital, is now associated with Dr. Halstead S. Hedges, Dr. Fletcher D. Woodward, Dr. G. S. Fitz-Hugh and Dr. F. J. Crigler at Charlottesville.

Dr. James F. Blades, having recently completed a residency in surgery at the Medical College of Virginia Hospital, announces the opening of his offices for the practice of general surgery in the Medical Arts Building and in the Medical College of Virginia Hospital, Richmond.

Dr. James T. Rountree, recently of Woodstock, has located with offices in the National Bank Building, Harrisonburg.

Dr. Alfred E. Powell, who recently completed an internship at the University of Virginia Hospital, has located for practice at Madison.

Dr. Dexter Davis, Roanoke, is taking special work at the Manhattan Eye, Ear and Throat Hospital, New York City.

Dr. Joseph E. Barrett, formerly Director of the Michigan State Hospital Commission and since 1939 Clinical Director of the Southwestern State Hospital, Marion, has been appointed Director of the program which the state hospital board has launched in planning a project of outpatient service.

Promotions in the faculty of the Medical College of Virginia, Richmond, are: Dr. Webster P. Barnes, to be Assistant Professor of Surgery; Dr. Guy W. Horsley, Assistant Professor of Surgery; Dr. Lawther J. Whitehead, Associate Professor of Radiology; and Dr. Rudolph C. Thomason, Assistant Professor of Ophthalmology.

DEATHS

Dr. Alex Doniphan Louthan, Wylliesburg, aged 71, died recently.

Dr. Stanley Haviland Martin, Richmond, aged 51, died recently of rheumatic heart disease.

WEST VIRGINIA

West Virginia Section of the Southeastern Surgical Congress held its annual meeting in Charleston, September 23, with Dr. R. H. Walker, Charleston, Chairman.

The following West Virginia physicians were awarded scholarships and attended the Southern Pediatric Seminar at Saluda, North Carolina, recently: Dr. H. H. Hancock, Union; Dr. E. A. Courrier, Keyser; Dr. L. E. Baron, Mannington; Dr. W. T. Smith, Glenville; and Dr. E. O. Vaughan, St. Albans.

Dr. Thomas W. Nale, Director of the Division of Maternal and Child Hygiene of the West Virginia State Health Department, has resigned to accept a position with the Carbide and Carbon Chemicals Corporation at its South Charleston plant.

West Virginia State Health Conference will be held at the Daniel Boone Hotel in Charleston, November 17 and 18.

West Virginia School for the Deaf and the Blind at Romney is making an effort to locate and bring into the schools all children between the ages of six and twenty-one years who do not have sufficient sight or hearing to enable them to be educated in the public free schools of the State. Inquiries should be made direct to Haven D. Umstott, Financial Secretary, West Virginia School for the Deaf and Blind, Romney, West Virginia.

Dr. Samuel W. Hamilton, of the U. S. Public Health Service, has been assigned to make a special study of West Virginia facilities for treating mentally defective.

Dr. U. C. Lovejoy, Omar, after taking a fellowship in gynecology and obstetrics at the Vanderbilt University Hospital, will be located in Nashville, Tennessee.

Dr. Benjamin I. Golden, Elkins, was elected Treasurer of the International College of Surgeons at the international assembly in Mexico City recently.

Dr. Howard Ross Crews, Pineville, and Miss Elizabeth Traylor Wilkinson, Huntington, daughter of Dr. and Mrs. R. J. Wilkinson, were married July 2.

Dr. Maurice J. Small, Parsons, and Miss Sylvia Cantor, of New Rochelle, New York, were married recently.

DEATHS

Dr. David R. Carden, Huntington, aged 72, died recently of pneumonia.

Dr. Irving Delbert Cole, Clarksburg, aged 60, died August 10.

Dr. Paul Adie Ford, Talcott, aged 50, died recently of perforated gastric ulcer.

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. . . can result from diets which are not quite good enough.[1] You know well, doctor, the insidious character of a sub-clinical vitamin and mineral deficiency. No pathology, no objective symptoms, no specific complaint — yet the patient feels below par, lacks vitality, and is often depressed. You take a rational step towards meeting this nutritional challenge with . . .

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Contains VITAMINS A, B_1, B_2(G), C, D, E and other B Complex factors, together with essential MINERALS: calcium, phosphorus, iron, copper, iodine, manganese, magnesium and zinc in Funk-Dubin balances.

There are 5 DISTINCT VI-SYNERAL* PRODUCTS supplying specially balanced vitamin-mineral potencies for FIVE DIFFERENT AGE GROUPS: 1. INFANTS AND CHILDREN 2. ADOLESCENTS 3. ADULTS 4. EXPECTANT AND NURSING MOTHERS AND 5. SPECIAL GROUP (MIDDLE-AGED AND AGED PATIENTS).

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[1] Report of National Nutrition Conference for Defense, Washington, D. C., 1941

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1930

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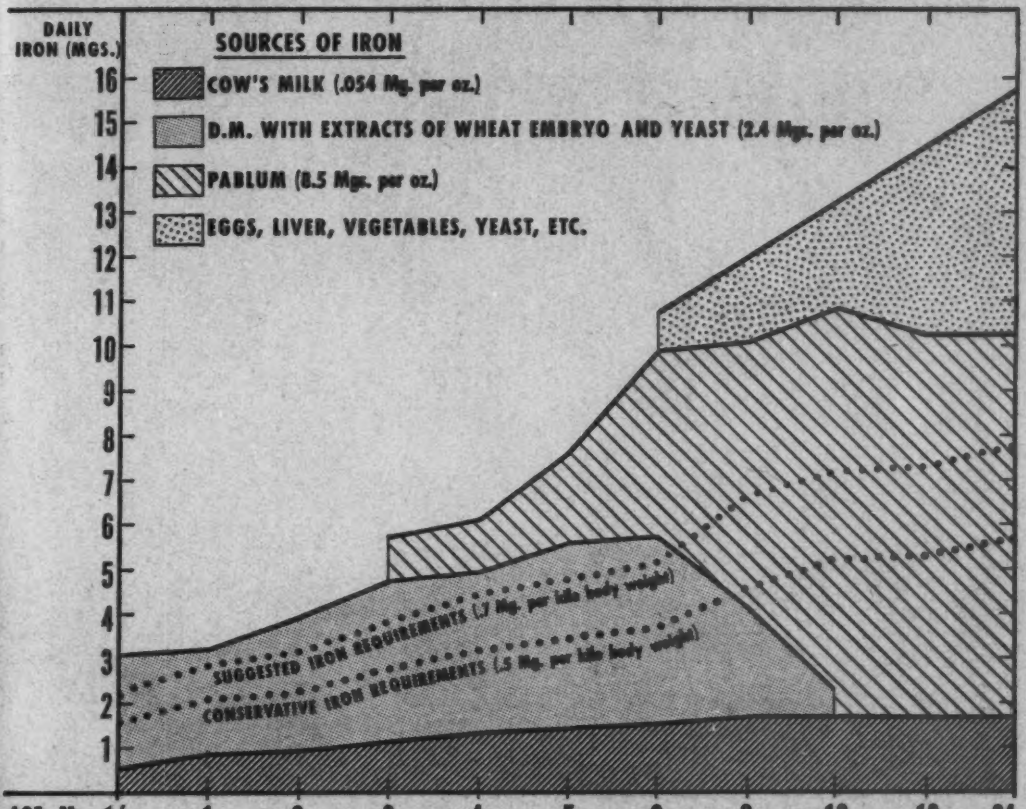
IRON REQUIREMENTS

DURING THE FIRST TWO YEARS



AGE, Mos.	1/4	1	2	3	4	5	6	9	12	18	24
WEIGHT, Lbs.	7	9	10	12	14	15	16	20	22	23	25
MILK, Oz.	10	16	18	21	25	26	28	32	32	32	32
D.M. WITH EXTRACTS OF WHEAT EMBRYO AND YEAST, Oz.	1	1	1¼	1½	1½	1¾	1¾	1	¼	0	0
PABLUM, Oz.	0	0	0	⅛	¼	¼	½	¾	1	1	1

During fetal life the infant accumulates iron in its body. After birth, this supply is rapidly depleted, the hemoglobin frequently dropping to 50% by the third month, especially in prematures. Neither breast milk nor cow's milk is capable of offsetting this loss, as they are deficient in iron. An infant requires one-half milligram of iron per kilogram of body weight. This chart shows that when the carbohydrate and cereal supplements contain iron, a sizeable margin of safety can be maintained, not only during the important first six months, but throughout the first two years of life.

The excess iron thus supplied over iron requirements averages close to 75%, and is needed because some iron is unutilized — a large amount in certain cases. In rapidly growing, or poorly nourished infants, and in the presence of infection, the need for iron may be greater than the chart shows; in some cases, periodic hemoglobin determinations may show the need for iron therapy.

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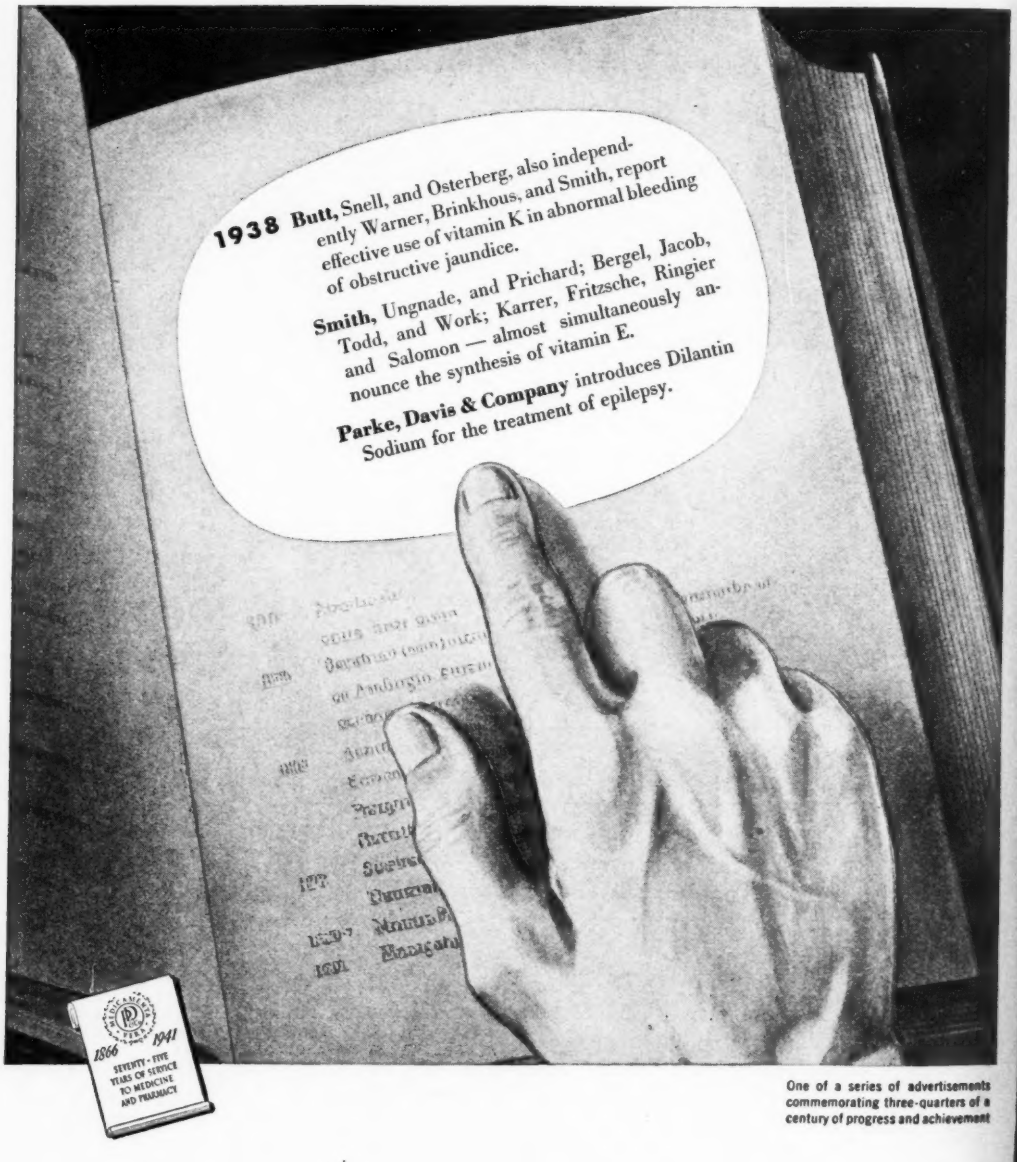
THESE NAMES, THESE YEARS...
HAVE HELPED MAKE MODERN MEDICAL HISTORY


1938 **Butt,** Snell, and Osterberg, also independently Warner, Brinkhous, and Smith, report effective use of vitamin K in abnormal bleeding of obstructive jaundice.
Smith, Ungnade, and Prichard; Bergel, Jacob, Todd, and Work; Karrer, Fritzsche, Ringier and Salomon — almost simultaneously announce the synthesis of vitamin E.
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